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A MANUAL

OF

DISEASES OF THE NERVOUS SYSTEM

BY

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CONSULTING PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL; PHYSICIAN TO THE NATIONAL HOSPITAL FOR THE PARALYZED AND EPILEPTIC

SECOND EDITION
REVISED AND ENLARGED

VOLUME I

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WITH ONE HUNDRED AND EIGHTY ILLUSTRATIONS, INCLUDING THREE HUNDRED AND SEVENTY FIGURES

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CORRECTIONS AND NOTE.

P. 184, line 10, *for* Fig. 57 *read* Fig. 64.
„ 191, „ 26, „ inflammation *read* information.
„ 207, „ 1, „ rectum *read* bladder.
„ 212, „ 24, „ p. 116 *read* p. 176.
„ 400, „ 26, „ puerperal *read* peripheral.

The reader is desired to append to the foot-notes on pp. 229 and 316 a reference to that on p. 552.

DISEASES OF THE NERVOUS SYSTEM.

INTRODUCTION.

CLASSIFICATION OF DISEASES OF THE NERVOUS SYSTEM.

A CLASSIFICATION of Diseases of the Nervous System, at once scientific, exact, and convenient for systematic description, is not at present possible. If we attempt to classify the diseases according to either seat or nature, we are met at once by difficulties which prevent any complete arrangement. These difficulties arise from the fact that many diseases affect more than one part of the nervous system, that the precise seat of other maladies is unknown, and that we are still ignorant of the nature of a considerable number of diseases. A rough division is often made into two broad classes of "organic" and "functional" disease. The first class, that of "organic diseases," comprehends those in which there is always a visible lesion, manifesting sometimes the morbid process which constitutes the disease, sometimes only the ultimate degree or result of that process. The second class, that of "functional diseases," is less definite, comprehending (1) those diseases that consist only in a disturbance of function, and are therefore properly so designated; and (2) many diseases which have this in common with true functional disease, that they are transient and not permanent, and that they are not known to depend on organic changes. But there is also a large class of diseases in which no constant lesion has been discovered, but which are not transient, and cannot well be placed in either of these classes. This simple classification is therefore inadequate. It may be well to consider in what respect it needs alteration to meet the requirements of our present knowledge.

The term "organic disease," as commonly used, means visible disease. But visibility is relative; it depends on the means of vision. The application of the microscope to pathology immediately increased the range of organic disease, and a similar increase has resulted, in our own day, from the use of staining agents, which render visible many changes that could not before be seen. Still it remains true that the degree of alteration determines visibility. Molecular changes in nutrition, considered as such, must be colossal to be detected. Such alterations, not sufficient to be seen, but still considerable, probably constitute the morbid process in many diseases that are commonly classed as "functional." Hence we may distinguish a class of "nutritional diseases" as to a large extent distinct from those that consist in a mere derangement of function. While distinguishing these two classes, we cannot entirely separate them; nutrition and function are mutually dependent, and prolonged functional disturbance must determine nutritional change.

Visible disease varies much in nature and degree. Some forms can always be seen with the unassisted eye; they change the shape or colour or consistence of the part. Other forms can be detected only with the aid of the microscope; while many may be seen with the unassisted eye when their degree is considerable, and only by means of the microscope in their early stage or when in slight degree. It is convenient to term such alterations, the visibility of which is altogether a matter of degree, "structural diseases." Thus we may distinguish four pathological classes of disease of the nervous system:

1. *Organic disease* (or "*coarse*" *organic disease*); such as tumour, hæmorrhage, softening.

2. *Structural disease;* such as most forms of sclerosis.

3. *Nutritional disease;* such as general paralysis of the insane, paralysis agitans.

4. *Functional disease;* such as reflex convulsions, and many forms of hysteria.

In the first class the morbid process always begins outside the nerve-elements themselves. In the second class it may begin in them or outside them. In the third and fourth classes these elements are probably always primarily affected.

Such a classification helps us to obtain clearer views of the primary relations of disease, but is not convenient for systematic description. For this a hybrid system is necessary, in which organic and structural diseases are classified according to their seat,—in the nerves, spinal cord, or brain. With each group may be placed some nutritional or functional maladies of local incidence. The bulk of these diseases are, however, best considered separately, after the organic diseases have been described, since many of them are of wide distribution or uncertain seat.

Classification has two purposes to serve. It serves to bring out the differences in the nature of disease, and thus to define our conceptions and make clear our distinctions. But this purpose is less obtrusive than the second,—to furnish us, as it were, with shelves on which to place our conceptions, and labels with which to ticket them, so that we may find them when we need, and study them in a useful order with the least loss of time.

Even if our knowledge were perfect it is not likely that any scientific classification, founded on natural distinctions, would serve the practical needs of an arrangement for *use*. Our present knowledge is unquestionably so very far from perfect as to be inadequate even for a complete natural classification; while an attempt to construct one of this character that shall be also of practical convenience is a mere waste of time. For use, we must divide where we ought only to distinguish; and we have to place near together, for convenience' sake, maladies that should be not only far from each other, but sometimes far from all others.

PART I.

GENERAL SYMPTOMATOLOGY.

SYMPTOMS AND THEIR INVESTIGATION.

THE nervous system is almost entirely inaccessible to direct observation. The exceptions to this are trifling: the termination of one nerve, the optic, can be seen; some of the nerve-trunks in the limbs can be felt either in the normal state or when enlarged by disease. As a rule, the state of the nervous system can be ascertained only by the manner in which its work is done, and morbid states reveal their presence by the derangement of function which they cause.

The functions of the nervous system are often divided into mind, motion, and sensation, but they are far more extensive than those words denote. The functions of all the organs of the body, all states of the blood-vessels and of the circulation within them, and even all processes of nutrition, are under the control of the nervous system. The disturbance of nerve-function produced by disease is conterminous in extent with the human organism. Our knowledge of these symptoms is constantly increasing in range and in degree. They differ, however, very much in relative importance. Some occur more frequently than others, and are more direct in significance. Many have a special relation to certain parts of the nervous system, while others occur in diseases of all parts. It may be well, at the outset, briefly to describe the latter—the general characters of some of their most frequent symptoms; while the more special symptoms can be more conveniently considered when we deal with diseases of that part.

MOTOR SYMPTOMS.*—Disturbance of motion is one of the most common effects of disease. Movement is produced by muscles, but the muscles are excited to contraction by the nervous system. Most alterations of motility, both defect and excess, are due to nerve derangement. The conditions are very few in which motor-changes originate in the muscles themselves. Defect of movement is of muscular origin only in some cases of primary alteration in the nutrition of the muscles. When there is a general impairment of nutrition, the muscles participate in this; they are ill-nourished, and therefore feeble. Such a condition is met with in chronic wasting diseases, in anæmia, and as an acute affection in all acute febrile diseases. This is the chief cause of the muscular weakness that so often succeeds fevers; it may even proceed to the degree of causing visible changes in the aspect of some fibres.

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* Some modification of the words kinesis, akinesis, &c., is sometimes used to designate motor symptoms. These words seem to me to add nothing to our knowledge or to its clearness—certainly nothing to compensate for their cumbersome character; and the definite addition all such words make to the knowledge that has to be acquired is not always sufficiently weighed against their supposed advantage.

In a few chronic diseases the muscles undergo a primary degeneration. One is the disease known as "pseudo-hypertrophic paralysis;" and allied is an affection in which there is atrophy of the muscles. Another is ossification of the muscles. In almost all other conditions, loss of power in the muscles is dependent on the state of the nervous system, either directly or through defective nutrition of the muscles, or both. The nutrition of the muscles depends on the motor nerves, although we are still ignorant of the precise nature of that dependence. The subject will be referred to again in the special account of the changes that result from injuries to the nerves.

So also with increased and involuntary muscular action—spasm. We know even less of what has been termed "idio-muscular contraction," that is, contraction originating in the muscle itself, than we do of the analogous paralysis. Whenever the whole of a muscle contracts, the contraction is probably due to the influence of the nervous system either directly causing the contraction or indirectly disposing to it. This is true even when, as in ordinary cramp, a contraction seems to us to be purely local. We can scarcely conceive that all the fibres of a muscle can contract simultaneously except under central influence. Even muscular "tone" is dependent on the connection of the muscle with the nerve-centre, since it ceases when the connection is destroyed. The partial contractions that are termed "fibrillary" are perhaps local, since they are so readily produced by mechanical stimulation, such as a tap on the muscle. But it is probable that fibrillary contractions, although sometimes local, are often of central origin. This is also true of the flickering contractions that are so common in persons whose nervous system is enfeebled, the "live flesh" of popular language. They may occur in any muscle, from the orbicularis palpebrarum (in which they are most common) to the gluteus maximus. After a lesion of the nerve-fibres to the muscle they occur in special degree; they are often extreme in the calf-muscles after sciatic neuritis.

Defect of movement may present every degree from slight weakness to absolute loss. There may be inability even to make the muscle contract, or the voluntary contraction may occur, but be too feeble to move the parts to which the muscle is attached. The latter difference is to some extent relative, since it varies with the readiness with which the part can be moved—i. e. with the resistance to be overcome. A contraction that would move a finger may be insufficient to move a leg. The difference may vary in the same part according to position. A degree of power in the flexors of the hip, that will move the leg when the patient is lying on his side, may be insufficient to do so when the patient is lying on the back. This difference is even more conspicuous in some other muscles; paralysis of the extensors of the elbow may be discovered only on an attempt to raise the hat: in other positions gravitation really effects that which the muscle seems to do. Sometimes the part is moved, but with less force than normal.

The term "paralysis" has long been applied to all degrees of defect of power, a distinction being made between complete and incomplete paralysis. Partial loss of power is also sometimes termed "paresis." The advantages of "paresis" over the word "weakness" are chiefly psychical, and depend on the popular horror of "paralysis," due to its application to absolute as well as to relative loss of power. The ideas of progress and incurability seem inseparably attached to the term. It is generally wisest to explain its medical use, and the fact that it has no significance as to the future.

Our means of measuring defects of muscular power are very imperfect. The force of some movements can be measured by means of an instrument called a "dynamometer." Several forms of dynamometer have been devised, but that in general use consists of an oval steel ring, which can be narrowed either by compression applied to its smaller diameter, or by traction applied to its longer diameter. The change in the shape of the dynamometer, and therefore the force exerted, is shown by the movement of an index on a scale that has a double graduation, a closer for compression, and a wider for traction. This form of dynamometer is known as "Charrière's," from the name of its first maker. An older form of dynamometer is that devised by Duchenne. It is less convenient for testing the grasp, although better adapted for indicating the force of traction. The use of these instruments is chiefly confined to the measurement of the grasp. The estimation of the strength of other movements is difficult, and liable to error, but with care a wider use of the instrument is practicable. In examining these it is best for the observer to oppose the movement and note the resistance that the patient can overcome. The wide variations in strength among different persons renders absolute estimation possible only when the defect is considerable. Slight changes are recognised by comparing the power on the two sides. It should be remembered that the limbs on the right side are normally stronger than those on the left, the relation being about as five to four. Care must be taken to secure uniformity of the conditions under which the observations are made.

The two movements that can be most conveniently measured by the dynamometer are the flexion of the fingers and of the hip. The former is estimated by the grasp; the latter by pressing the raised leg down by means of the dynamometer placed over the end of the femur above the knee. The person tested being seated on a chair, the knee flexed, and the foot raised from the ground, I find that the force needed to overcome the contraction of the flexors of the hip is nearly the same as that of the grasp of the hand in the same person and on the same side.

Inco-ordination of Movement.—By this term is meant a defect in the co-ordination of muscular contractions by which a movement is produced. Every movement is due to the contraction of a series of fibres, which seldom corresponds to the series massed together in a "muscle." Fibres, not muscles, are represented in the structure of the brain, and those that cause a simple movement may be in several muscles. The *synergic* action of these in a definite degree, and at a certain time, is essential. So also is the simultaneous contraction of the opponents of these acting muscles, which are thus steadied; this *antergic*[*] contraction is essential for the exertion of force. Synergic and antergic contractions seem to be directly proportioned to the force exerted; but the antergic is in inverse degree to the movement. Defect in antergy not only does not interfere with *movement*, but prevents the exertion of force by permitting an excess of movement.

But it is not customary to apply the term "inco-ordination" to any derangement of movement except that which is irregular in time. The variations in the time of the muscular contractions makes the form of movement variable and irregular. This irregularity is also called "ataxy."

A method of estimating its degree consists in making the patient suddenly try to touch, with the point of a pencil, a spot on a sheet of paper suspended before him, his eyes being closed at the moment of the attempt. After a certain number of attempts the average distance from the spot of the dots he has made is ascertained, and, compared with the result given by a normal individual, affords a numerical indication of the degree of inco-ordination. The method is effective, although the result is perhaps scarcely worth the trouble.[†]

Whenever there is inco-ordination, spontaneous movements occur if the patient tries to hold the limb in a certain posture; the varying degree of contraction deranges the fixation as well as the movement of the limb: deranged fixation means spontaneous movement, co-ordination being essential for steadiness of uniform contraction, as well as for the steadiness of the varying contractions of movement. These involuntary movements constitute a delicate test for the presence of ataxy.

It is necessary to distinguish co-ordination and the *direction* of movement to a definite end. The motor process for the latter depends on true sensory guidance, chiefly on the senses of touch and sight. It thus depends on peripheral guidance. A sempstress with loss of sensation in the fingers cannot sew unless she constantly watches them. A compositor, whose sense of touch is dulled upon the tips of thumb and index finger, cannot pick up the type, or if he does pick it up, drops it again, unless he constantly watches the movement. Strictly, this is included in the idea of co-ordination, but its loss is not spoken of as inco-ordination. There is also a peripheral guidance from the muscles, but this is related to co-ordination proper, rather than to the direction of movement, and it will be more conveniently considered in connection with the functions of the spinal cord. Indeed, the mechanism of co-ordination, and the defect in the elements of the process that cause inco-ordination, can only be discussed in connection with those functions.

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[*] I use this term because we have none to express antagonistic co-operation. This is synchronous with that of the acting muscle, but not synergic.

[†] Blix, 'Neur. Centralb.,' 1884, p. 83.

Sensory Symptoms.—Deranged sensation is another very common symptom. It has often to be searched for, as a significant defect may be unknown to the patient. Each form of sensation, touch, temperature, and pain, must be separately tested, since one may be affected and not another.

Sensibility to Touch.—The structures that subserve tactile sensibility are stimulated chiefly by motion—by change in contact. Prolonged contact, uniform in its degree and place, is perceived but little, and only by close attention. It is the change that excites an impulse. In testing tactile sensibility care must be taken that the instrument employed does not give an impression of heat or cold, lest the patient perceive by the sense of temperature, that which he cannot perceive by the sense of touch. The observer's finger may be used, if it is not cold, or the skin may be touched with the feather-end of a quill, a piece of string, &c. The eyes of the patient should be closed during the examination, and, since he is apt to fancy that he feels a light touch when he does not, he should occasionally be asked if he can feel, when no contact is made. Loss of sensibility to touch is termed "anæsthesia," but this word is often applied loosely to all forms of impaired sensibility.

In examining the tactile sensibility, it is important to ascertain, not only whether the patient can feel, but whether he is able to recognise the place touched,—whether he can correctly "localise" the sensation. For this he must be asked, not only whether he feels the touch, but also to say or point out where he feels it. The part touched should be frequently varied. Another test for tactile sensibility depends on the fact, ascertained by E. H. Weber, that the distance apart at which two points are discriminated is nearly the same in different individuals in the same part of the body, although it varies much in different parts of the body. The normal distance being known, the increased distance at which the points have to be placed for their discrimination is a measure of the degree of defect. The points should not be so sharp as to occasion pain. Common compasses may be employed, but the most convenient "æsthesiometer" is one contrived by Sieveking, in which the compass points are attached to a graduated bar. The greater the degree of tactile sensibility, the nearer together can the points be, and still be discriminated. The distance at which they are normally distinguished must be known before any inference can be drawn. The most important average distances, ascertained by Weber, are as follows: the distance is smallest, *i. e.* the sensitiveness is greatest, at the tip of the tongue, where the points are discriminated when only 1·5 mm. apart. Then come, in order of sensitiveness, the finger-tips, 2 to 3 mm.; the lips, 4 to 5 mm.; the tip of the nose, 6 mm.; the cheeks and the backs of the fingers, about 12 mm.; the forehead, 22 mm.; the neck, 34 mm.; the forearm, lower leg, and back of foot, 40 mm.; the chest, 45 mm.; the back, 60 mm.; the upper arm and thigh, 75 mm. Slight variations exist in different individuals, and a deviation from the normal that is uniform in degree throughout the body is probably physiological. In using this test it is necessary to touch the skin with the two points at the same moment, and with equal pressure. The examination requires time and patience, and the results are considerably modified by the intelligence of the patient. Moreover, the power of discrimination is increased by practice. It is rare to obtain a conclusive result unless there is a degree of defect that causes a slight absolute loss, such as may be detected with a feather. For these reasons, the practical value of the test is less than was at first anticipated. It is most useful for the estimation of changes of sensibility in the same person and the same part.

Another test that has been proposed is to move a pointed object along the skin, and note the minimum movement that can be recognised as such (Leube). This test has not come into general use.

Curious modifications of tactile sensibility are sometimes observed. In one of these, a single touch is felt as if it were two or even three, a condition that has been termed "polyæsthesia" (Fischer). In another, an impression on one part is referred to some other part, usually in the same limb, rarely in another limb.* In another variety, an impression on one part has been referred to the corresponding place on the opposite side of the body; this has been termed "allocheiria" (Obersteiner).

Perception of the degree of pressure on the skin is probably subserved by the nerves for tactile sensibility. It may be estimated by simple pressure applied through any instrument, or by small weights, or by an instrument contrived for measuring the pressure applied to an artery to ascertain the tension of the pulse. In this, a spring indicates the degree of pressure applied. The part tested must be supported, so that the muscles are not brought into action. The point to be ascertained is the minimum variation that can be recognised. In health, this is about one twentieth of the total pressure, whatever the latter may be (Weber).

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* In most persons there are spots in the skin whence a painful sensation seems to dart to a distant place; *e.g.* a prick on the thigh in a limited area may be felt also as a sharp pain near the scapula. This shows how structural connections must exist favouring the "reference" of sensation to a distance from the nerve stimulated. Another instance of the same perverted reference is presented by the frequent neuralgic pain in one jaw due to a carious tooth in the other jaw, which seems painless.

Sensibility to pain is subserved by what are called nerves of "common sensibility." It may be tested by a prick or a pinch. For a prick, too fine a point must not be used, not only because a sharp point may penetrate the skin, but because, in the less sensitive parts of the skin, where the terminal nerve-plexus is wide, a fine point may here be unfelt, although felt readily at a place close by. Nothing answers better than the point of a quill pen. The faradic current may be employed to test sensibility to pain, wire terminals being most suitable for the purpose. Its advantage is the delicacy with which it can be graduated, but it does not furnish any absolute standard, and needs more study before it can be usefully employed.

Sensibility to pain may be changed with or without sensibility to touch. Its loss is termed "analgesia," but is often included in the general term "anæsthesia." A painful sensation is often felt more acutely than normal; this is called "hyperæsthesia," or, more correctly, "hyperalgesia." Occasionally a touch on the skin gives rise to pain, but it is probable that this is due to the stimulation of the over-sensitive nerves of common sensibility, and is not an intensification of a tactile sensation. Both tactile and painful impressions may produce sensations that are abnormal in character, described as "thrilling," "tingling," &c. This perverted sensation has been termed "paræsthesia," or "dysæsthesia," words that have also been applied to purely subjective sensations.

Sensibility to temperature is usually affected with sensibility to pain, but often not in the same degree, and one may be impaired without the other. There may be an absolute inability to recognise either heat or cold as such, or slight degrees of each may be unperceived while considerable degrees are recognised correctly. In the latter case there is impairment of the power of perceiving differences in temperature, analogous to the defect in the perception of differences in pressure. There may be observed sensation whereby hot objects feel cold, and cold objects hot. The pain that extreme degrees of temperature normally produce may be felt with undue readiness, or less readily han normal.* When the interval that elapses before a sensation of pain is felt is increased, so also is the temperature; if sensation of pain is delayed, that of temperature may be delayed also. It must be remembered that, in health, this sensation is slower than that of pain, because time is required to raise the temperature of the skin sufficiently to stimulate its nerves. For coarse examination, hot and cold spoons may be employed, or a hinged tongue depressor, one half of which is warmed. Both heat and cold must be sufficient in degree to be unquestionable, so that hesitation in a patient is certainly subjective, and not due to equivocal "lukewarmness." For ascertaining the power of differential discrimination, test-tubes, containing water at different temperatures, are necessary. For practical purposes the result thus obtained is hardly commensurate with the time required for the examination.

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* See on this point Donath, 'Arch. f. Psych.,' Bd. xv.

It is always necessary to test separately the sensibility to heat and to cold, as there is strong reason to believe that they are subserved by different nerves. If very minute points of the skin are examined, it is found that at some points only heat, at others only cold, is perceived. The stimulation of points of the skin by electricity shows also that at some points the peculiar electrical sensation is produced, at others sensation of cold, at others sensation of heat. Further, the points identified as "cold points" and "warm points" by the one method correspond with the points ascertained by the other method.* The conclusion from these observations is confirmed by the fact that in disease the sensibility to heat and to cold may be affected in different degrees.

Muscular Sensibility.—The term "muscular sense," as often used, includes more than one form of sensation. There is, first, a true muscular sensibility. The muscles are abundantly supplied with sensory nerves, which are said to end in the interstitial tissue between the muscular fibres (Tschirjew). They seem to be stimulated by pressure and traumatic processes, like other sensory nerves, and also in a special way—by traction on the muscle. Thus a squeeze of the calf causes a muscular sensation of an undeniable quality, and strong passive flexion of the foot on the leg produces a sensation which is distinctly produced *in* the muscle. They are, moreover, strongly stimulated when the fibres are shortened by extreme contraction, which causes the most acute pain that is ever felt in the muscles. It must be ascribed to the compression of these nerve-endings, since apparently no afferent impressions come from within the fibres. It is thus a proof of their susceptibility to pressure-excitation. After cramp, the nerves are left in a state of such exalted excitability that slight extension of the muscle gives rise to pain, although such extension has no sensory effect in the normal state. Thus these two mechanical influences are those to which these nerves are chiefly sensitive. The muscular pain caused by electrical stimulation, as distinct from the pain felt in the skin, may be due to the stimula-

* Blix, 'Zeitschr. f. Biologie,' Bd. xx, p. 141; Eulenberg, 'Zeitschr. f. klin. Med.,' Bd. ix, Heft ii. The observations have also been corroborated by Goldscheider and Herzen.

tion of these nerves, chiefly direct. But these sensations of pain, although due to "muscular sensibility," are not included in the term "muscular sense." They are, however, of great importance on account of the conclusive evidence they afford of the mode in which these nerves can be stimulated.

The chief knowledge that is ascribed to the "muscular sense" is that of the character of movement and posture due to the action of the muscles, and also of the character of passive movement (by an external force) and of posture when the muscles are at rest. It is evident, however, that here we have two very different conditions—in one of which there is activity of all the motor elements, while in the other these are at rest. Without motor action, there can be only the sensory action that never ceases; in active states this continues, but motor action is superadded. Hence, these two elements that are comprehended in the term must be separately considered.

Strange to say, the problem that appears the more complex receives most elucidation from certain facts. We have almost direct evidence of the source of our knowledge of active posture and movement, while we have only indirect evidence of the origin of our knowledge of passive movement and rest-posture.

The evidence shows that the former is due, at least in great measure, to the effect on consciousness of the activity of the cells causing the movement. The proof of this consists in two facts. (1) In palsy of an ocular muscle, objects seen are referred to the position (in relation to the body) that they would occupy if the movement corresponded to the innervation; it is the latter, i.e. the activity of the nerve-cells, to which the perception of movement corresponds. (2) After amputation of a limb, a person who makes an effort to move the lost part, seems to feel the part move just as if he did move it. These facts show that the motor process is the chief source of our knowledge. But, in the case of such a phenomenon, we are not justified in assuming that the source of the chief is the source of the whole. The perception is not so well defined as to permit us to say that it may not in some minor degree be the result of the influence about to be described, which may co-operate with the sense of active innervation, although we may be unable to discern the difference that it makes in the total result. Indeed, if the following reasoning is valid, such co-operation must be admitted.

Our knowledge of rest-posture and passive movement must be derived from in-coming impulses. No other source for it is possible. The afferent impulses to which the knowledge is due are not those from the skin; the sense of posture may be lost when cutaneous sensibility is normal; and conversely, the skin may have lost most of its sensitiveness when this knowledge is perfect. These two facts are illustrated by unilateral lesions of the spinal cord, as well as by other symptoms of disease. The source of these perceptions must therefore be the deeper nerves, those of the muscles and joints, and chiefly of the former. When the subject of central co-ordination is discussed, evidence will be adduced to show that those afferent impulses from the muscles pass to the cerebellum and act through it on the motor cells of the cerebral cortex, regulating their state and controlling their activity, the continuous slighter activity of " rest," and the occasional greater activity that the will induces. In this influence on the cells (or rather in the effect it produces), we have a source for these perceptions of passive states. We are thus led to refer our perceptions of passive posture to the same cells as give rise to our perceptions of active posture. Our knowledge is the result of perception and not of sensation proper;—the impulses that cause our perception of passive states are not directly *felt*. That these impulses also enter into our perception of active states, completing them, and rendering them perfect, is highly probable.

Our recognition of resistance to contraction affords one of the most delicate tests for "muscular sensibility." It is used by testing the ability to detect differences of weight, *i. e.* variations in the resistance to contraction. In this examination the patient's eyes should be closed, and the objects used should be of uniform size. Leather balls like small cricket balls, containing various weights from two drachms to two pounds, are in use at the Queen Square Hospital for this purpose. It is desirable to eliminate as far as possible the stimulation of the cutaneous nerves. This may be effected in three ways: (1) Reducing it to a minimum area, placing the weights in a bag suspended by a string to the part to be tested, so that only a small area of the skin shall be pressed upon; (2) by increasing it in area (Hitzig), so as to lessen the pressure in any given area such as is pressed upon in the ordinary way; (3) by increasing the pressure in both area and degree, so that the addition affected by the weight shall bear only a small proportion to the total stimulation. This may be done by attaching the weight to the lower of two bands, one above the other, below the part of the limb to be tested.

(1) The point to be tested is the power of discriminating weight. This is greater than in the case of the nerves of the skin, since a difference of one fortieth of the total weight can be recognised under normal conditions. (2) The sensitiveness of the muscles to pressure and traction. (3) Their sensitiveness to electrical stimulation. It is difficult to test accurately the electro-sensibility unless the sensitiveness of the skin is removed, as by the injection of cocain.

The power of recognising the posture into which the limb is put is also regarded as a test for muscular sensibility; if cutaneous sensibility is normal the parts handled must be grasped firmly, and pressed on both sides, so that the direction of pressure may not suggest the posture. Several observations should be made to eliminate the chance of accidental error.

REFLEX ACTION.—Many symptoms of disease of the nervous system are due to derangement of the various reflex actions. These are numerous, and we can now only consider their general characters. Each action is effected through an afferent sensory nerve, an efferent motor nerve, and a system of nerve-cells and fibres connecting the two in the spinal cord, the "reflex centre." The centre is usually a complex and often extensive structure, and in it are paths of different "resistance," determining the form of reflex action and its extent, according to the source and intensity of the sensory impression. The reflex centre is between the roots of the sensory and motor nerves concerned, and with them constitutes what may be termed a "reflex arc." The sensory impulse may not only excite a motor process, it may also pass up to the brain, and effect consciousness as a sensation. It is probable that these are subserved by the same nerve-fibres, so far as the skin nerves are concerned, but by different fibres from the muscles. Further, the reflex centre of the spinal cord is to some extent under the control of encephalic centres—a point of very great importance. Two chief forms of reflex action must be distinguished. The first is that excited chiefly by stimulation of the skin; the second is that which is excited by a peculiar mode of stimulation of deeper nerves, probably only those of the muscles. The latter is obtained, among other ways, by tapping a tendon, and therefore was at first, and still is sometimes, called "tendon-reflex action"—a most undesirable name.

Cutaneous Reflex Action.—The reflex movements obtained from the skin are excited either by a gentle stimulation, as a touch, than by a strong, painful impression. They consist of a single movement in most instances, either quick or slow, but often somewhat complex, and extensive in proportion to the degree of the stimulation effected, and to the character of the sensory stimulation. Often muscles at a distance are influenced by a powerful stimulation, either with others or alone. A strong impression may cause a reflex action so wide as to involve most of the muscles of the body. As a rule a painful impression causes a quick flexion movement, as if for escape. Thus a prick near the knee causes a reflex flexion of the hip. On the other hand, a tactile impression causes a much more complex and irregular movement.

The slighter degrees of excitation cause a contraction in the muscles close to the part of the skin that is stimulated, the impulse passing, in the spinal cord, only to the related motor centres. This limitation of the process renders them important in diagnosis, as will be shown in the chapter on the functions of the spinal cord, where they are more fully described. The cutaneous reflex actions may be excited at almost any part of the skin, but at some parts they are very definite in character, and are distinguished by special names. The most important are the "plantar reflex," from the sole; the "gluteal reflex," a contraction in the gluteus when the skin over the muscle is stimulated; the "cremaster reflex," a retraction of the testicle on stimulation of the skin on the inner part of the thigh; the "abdominal reflex," in the muscles of the abdominal wall when the skin over the side of the abdomen is stroked; the upper part of this reflex is a very definite contraction at the epigastrium, and has been termed the "epigastric reflex." A series of reflex actions may be obtained in the muscles of the back, the highest being in the muscles of the scapula. Others may be and have been distinguished and named, but this process may easily be carried too far. These spinal reflex actions vary in their excitability in different individuals, and are always more readily produced in the young than in the old. In the region of the cranial nerves the most important reflexes are those of the eye—(1) the conjunctival reflex, (2) the contraction of the iris on exposure of the eye to light, and its dilation on stimulation of the skin of the neck.

Muscle Reflex Action; "Tendon-reflexes."—The second group of phenomena which depend on reflex action were first systematically studied by Erb and Westphal. All occur in muscles that are in a state of slight tension, and are produced by a sudden increase of this tension or by some other sudden mechanical influence, but voluntary contraction superadded to passive tension diminishes or prevents them.*

The first of these is the jerk of the leg which occurs when the patellar tendon is tapped. It has been called the "knee-phenomenon" by Westphal, the "patellar tendon-reflex" by Erb, the "knee-jerk" by myself. To obtain the jerk, the knee must be flexed so that the quadriceps femoris is gently extended, and the leg must be free to move. If then the patellar tendon is struck, the quadriceps contracts and jerks the leg forwards. The most convenient position is with the knee to be tested flexed nearly, but not quite, at a right angle. The posture commonly employed is with the leg to be tested across the other, the knee of the supporting leg being at a right angle (Fig. 1). But if the leg to be tested is stout, its tension in this position may be too great to permit of any movement. In such case the observer may place his arm beneath the patient's thigh, just above the knee, and rest his hand on the patient's other knee (Fig. 2). Children may sit on the edge of a chair, adults on the edge of a table. If the legs are vertical, the effect of the recoil and of the muscular contraction must be carefully distinguished. If the bent fingers of each hand are interlocked, and the hands pulled strongly, the knee-jerk occurs more readily (Jendrassik). It is essential that both sets of muscles should be free from voluntary contraction. Any contraction of the flexors may be ascertained by feeling the hamstring tendons, and pressure on these by the fingers often helps to secure relaxation. The blow may be given by the side of the hand, a percussion hammer (Fig. 2), or a stethoscope with an india-rubber edge to the ear-piece. If the jerk is doubtful, the skin should be bared. In many cases the movement may be obtained by a downward blow upon the patella, by a blow on the quadriceps tendon above the patella, or by a blow on the substance of the muscle—in fact, by any mechanical stimulation that can reach the muscle—almost as readily and strongly as by a blow on the patellar tendon. When it is in excess, it may be conveniently brought out in a somewhat different way. As the patient lies in bed, the finger of one hand is placed across the quadriceps tendon just *above* the patella, and the patella pushed down, so as to make the quadriceps tense. The finger is then percussed in the direction in which the patella is being pushed, so as suddenly to increase the tension in the muscle. The blow is instantly followed by a contraction, jerking the patella and finger upwards. Very often this single contraction is immediately succeeded by a second, and this by a third, and so on—a series of quick clonic contractions, or "clonus," recurring as frequently as eight per second. By grasping the patella firmly, and suddenly pushing it downwards, so as to make the muscle tense, this clonus may also be set up. It may continue as long as tension is kept up, but instantly ceases when the muscle is relaxed.

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* This is not in harmony with the results of the experiments of Weir Mitchell; but his facts are inadequate to establish his statement. The conditions that influence the phenomena, and the effects of these, are very complex.

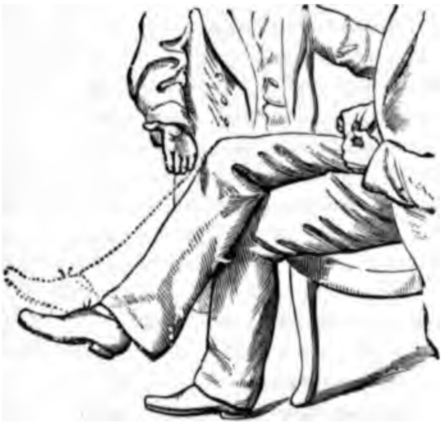

Fig. 1.—The Knee-Jerk.
The dotted line indicates the movement which follows the blow on the patellar tendon.

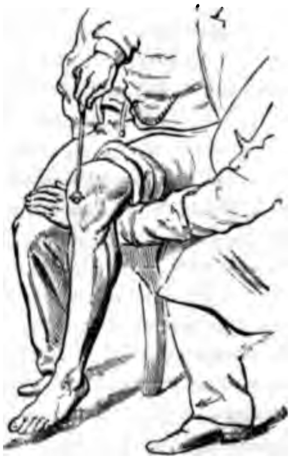

Fig. 2.—The Knee-Jerk.
Method of obtaining it when it is not readily produced in the ordinary way.

The next important phenomenon belonging to this group occurs at the ankle-joint. If the calf-muscles, which are connected with the Achilles tendon, are made tense, and this tendon is tapped, the muscles contract, causing a slight extension movement of the foot; just as the muscles of the thigh contract when the patellar tendon is struck. In cases in which these phenomena are excessive,—just as sudden tension in the thigh-muscles will cause a contraction, followed by others in a continuous series—so, in such cases, if the calf-muscles, which extend the ankle-joint, are suddenly put on the stretch by pressing the hand against the sole of the foot (Fig. 3), a quick contraction occurs, instantly ceasing, but if the pressure is kept up, instantly renewed, and recurring as long as the tension is maintained, as a clonic series of spasmodic contractions—the "ankle-clonus" or "foot-clonus" (or "foot-phenomenon"— Westphal). It can often be obtained best when the knee is not completely extended. The movement is very uniform under the same conditions, varying from six to nine contractions per second. By attaching a writing point to the foot, and making it trace a line on a revolving cylinder covered with blackened paper, tracings may be obtained (Fig. 4), which are almost as regular as those of a tuning-fork. The clonus in the extensors of the knee has the same time and is evidently of the same nature. A clonus quite similar may sometimes be obtained in other muscles—peronei, flexor brevis pollicis, flexors of the fingers, &c.

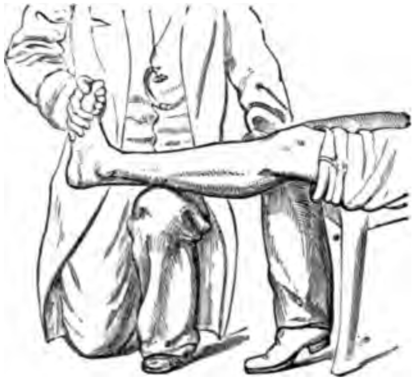

FIG. 3.—METHOD OF ELICITING THE FOOT-CLONUS.

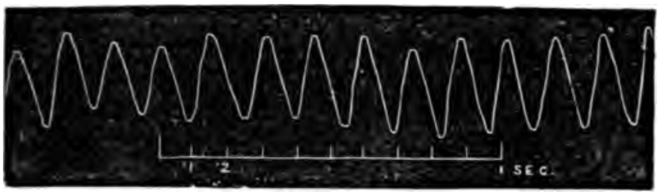

FIG. 4.—TRACING OF THE FOOT-CLONUS IN PARAPLEGIA.
(The tracing reads from right to left.)

When a tendon is tapped, and its muscle contracts, the occurrence has so much the aspect of a true reflex action that it was assumed by most investigators to be such, the stimulus being the excitation of nerves in the tendon. This view received apparent confirmation by the discovery of certain facts: (1) That there are nerves in tendon. (2) That these phenomena depend for their occurrence on the integrity of the reflex path to, through, and from the spinal cord, and are arrested by a lesion in this path. By experiments on animals (in whom similar contractions may be obtained) it has been found that they are prevented by division of the nerves to the muscles, by division of either the anterior or posterior roots of the spinal nerves, or by destruction of the spinal cord.* The knee-jerk cannot be obtained in locomotor ataxy (damage to the posterior nerve-roots), or in infantile paralysis (damage to the anterior grey matter, part of the reflex centre). (3) That these phenomena are increased in some cases, together with the reflex action from the skin.

These facts certainly prove that some reflex influence is concerned in the production of the phenomena. But (as Westphal has always maintained) they do not necessarily prove that the contractions depend on a simple reflex action, and it is certain that they do not depend on a reflex action from the tendons. Passive tension is necessary for the tap on the tendon to be effective. When the phenomena are in excess, sudden tension alone will suffice to develope the contraction. Tension acts upon the muscle as well as upon the tendon, and frequently the contraction may be distinctly excited by stimuli which act on the muscle and have no action on the tendon. For instance, in cases in which these phenomena are in excess, if the foot be gently pressed up so as to make the calf-muscles tense, and the muscles on the front of the leg be tapped, the calf-muscles contract (just as they do when, under the same circumstances, the tendon is tapped), and cause a brief extension movement of the foot. I have termed this the "front-tap contraction." It is a very delicate test of increased irritability, and it is also of considerable theoretical interest, since we have in it a contraction developed by a stimulus which does not in any way affect the tendon. It can affect the gastrocnemius directly, for by placing the hand on the calf, a vibration may be felt through the leg. If the tibia, instead of the muscle, is tapped, the contraction is much slighter, or does not occur. Moreover, a tap on the tendon itself *only excites the contraction when it increases the tension of the tendon;* i. e. *when it acts upon the muscle also.* In the case of the Achilles tendon, this may be easily demonstrated with a little care. A gentle tap on the side of the tendon will excite the contraction as readily as a tap on the back of the tendon, but if the outer edge of the tendon is supported (as by the fingers of an assistant), the same tap will no longer be effective, because it no longer increases the tension. The strongest proof, however, of the independence of the phenomena on any stimulation of the tendon, is afforded by the experiments of Tschirjew, who divided carefully all the nerves to the patellar tendon, and still found that the tap upon it made the [illegible] muscle contract.

Thus the evidence seems conclusive that the contractions are not excited by stimulation of the nerves of the tendon, but that the [illegible] originates in the muscle, the tendon being only, so to speak, [illegible] by which that stimulation is produced. But if the [illegible] is stimulated and then contracts, is not the contraction excited [illegible] as Westphal has, from the first, urged? A reflex action takes [illegible] which is needed for the stimulus to travel to and from [illegible] for the reflex process to occur in the centre. According to received physiological data, an interval of at least one fifteenth of a second would be needed for the knee-jerk, if it were a reflex process, and rather more for the movement at the ankle. I have found that when the Achilles tendon, or the front of the leg is tapped, the resulting contraction occurs in about one thirtieth of a second.* The interval for the knee-jerk has been found to be about one twenty-fifth or one thirtieth of a second.† If the patella is pressed down and tapped, in the way I have just described, I have found that the interval between the tap and the resulting contraction is often not more than one fortieth of a second. The shortness of the interval makes it less easy to believe that these contractions are reflex, than that they are excited locally.

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* [illegible] Tschirjew, 'Archiv für Psychiatrie,' Bd. viii. Heft 3.

At the same time, too much stress must not be laid on time measurements. It is certain that some reflex process is concerned in the phenomena. If we regard the contractions as local, we have still to account for the irritability which permits the local stimulus to cause a contraction. This irritability is developed by passive tension. If the muscle is relaxed, the fibres may contract if they are struck directly, just as do the fibres of a separated frog's muscle, but no contraction can be produced by striking the tendon. Hence I have suggested that the tension excites, by a reflex influence, a state of irritability to local mechanical stimulation,—such as that of a tap on the muscle, on its tendon, or even the vibration from a tap on the bone or on adjacent parts. But only that form of mechanical stimulation is effective which *suddenly* increases the previous tension. It is only because the tap on the tendon does this so readily, that the tendon is the means by which the contractions are most easily produced, and through which they have been chiefly studied and prematurely named. If the tension put on a muscle is gentle and gradual, it may only develope the irritability, and an additional local stimulation is necessary to produce a visible contraction.‡ If, however, the tension is sudden and forcible, it not only developes the irritability, but produces visible contraction in the muscle thus rendered irritable —as in setting up the foot-clonus. I have shown that the relaxation of the muscle, between the successive contractions, is not complete: there is a persistent residual contraction, *i. e.* a tonic contraction on which the clonic contractions occur. When one clonic contraction is over, the tension continuing, a second is instantly developed.

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* 'Med.-Chir. Trans.,' 1879, p. 292. The measurement has been since confirmed by Waller, 'Brain,' July, 1880.

† ·039 sec. Burckhardt; ·032—·034 sec. Tschirjew; ·04 sec. Brissaud; ·03—·04 sec. Waller; ·03 sec. Eulenberg. Some measurements which I made of the interval ('Med.-Chir. Trans.,' 1879, p. 275) gave a longer interval, probably in consequence of the movement of the foot being taken as the indication of the commencing contraction. "Load" will increase greatly the period of latent stimulation, probably by causing the initial contraction to expend itself on the elasticity of the muscle. The measurements given above were obtained by recording the commencing contraction of the muscle.

‡ This explanation was originally given for the "foot-phenomenon" alone ('Med.-Chir. Trans.,' 1879, p. 295). I now think that it is equally applicable to the knee-jerk. The evidence of the identity in nature of the two is very forcibly stated by Waller (loc. cit.).

The production of the clonus seems thus to depend on the existence of a law limiting the time of muscular contraction. If a fibre cannot continue contracted, it relaxes and re-contracts. This is so in muscular "tetanus," although the alternation in different fibres prevents its manifestation. But in the effect of sudden tension at the same instant, both contraction and relaxation occur in all the fibres at the same time, because all are stimulated alike and at once. We apparently have an example of the "single muscular contraction" such as physiologists obtain by an induction shock.* It agrees with this in character and time. We have no facts to suggest that such contractions could be produced through a reflex mechanism.

The sensory nerves of muscles have been shown by Tschirjew to commence, not within the muscular fibres, but in the interstitial connective tissue. The afferent impulse produced by tension is apparently due to the tension acting on these nerves: the visible contraction is excited by tension or vibration affecting the muscular fibres themselves. The latter is ineffective unless the muscles are brought into a state of special excitability through the cord. The reality of an afferent impulse from the muscle, produced by tension, is demonstrable to anyone who will permit his ankle to be suddenly flexed. A distinct pain is felt in the muscle (none, be it observed, in the tendon). It is not surprising, therefore, that this afferent impulse should, very often, not merely develope the reflex excitability or tonic contraction, but also cause a more widely-spread reflex action. The attempt to get the foot-clonus, for instance, will cause a flexion of the hip-joint; the attempt to obtain the knee-jerk may cause a movement in the opposite leg or a start back of the body. But these reflex contractions, if carefully observed, may be seen to *succeed*, at an appreciable interval, the immediate contraction. Burckhardt has found that the latent interval for a skin-reflex is three times as great as for the knee-jerk. This theory of reflex irritability and local stimulation affords a full explanation of all the relations of these phenomena, and it is the only theory which does so.

It seems, therefore, that the term "tendon-reflex" is altogether inaccurate. The phenomena are, according to the explanation above given, dependent on a "muscle-reflex" irritability, which has nothing to do with the tendons. If each contraction is reflex, it must be excited by an afferent impulse from the muscle-nerves, not from tendon-nerves. This fact seems conclusive and inevitable. Hence if we wish to describe them by a general term, it is best to employ one which does not involve any special theory of their nature. They have been called "tendo-muscular phenomena," but the intervention of [illegible] tendons is not necessary for their production; the one condition which all have in common is that passive tension is essential for their occurrence, and I have suggested[*] that they be termed *myotatic* contractions (τατικός, extended). The irritability, on which they depend, is due to and demonstrative of a muscle-reflex action which depends on the spinal cord. It is highly probable, as Tschirjew suggested, that the condition on which the myotatic irritability depends, is identical with muscular "tone." Since the experiments of Heidenhain, it has been generally admitted that "tone" depends on tension, and is a reflex process.[†]

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* See Foster's 'Physiology,' 1888, pt. i, p. 78.

A true "tendon-reflex" may be excited by pinching the tendon, but this is a start of the whole limb, precisely such as results from a pinch of the skin. It is an instance of the pain-reflex described above. The importance of discarding the tendon-reflex theory (and the name that must always involve it to those who have not studied the matter) is very great, for it prevents the comprehension of the true nature of other phenomena of great importance.

Paradoxical Muscular Contraction.—This name has been given by Westphal[‡] to a slow tonic contraction occurring in a muscle when its attachments are suddenly brought nearer. It is best seen in the tibialis anticus; if the foot is grasped, and passively flexed on the leg, the tibialis anticus contracts, sometimes after an appreciable interval; its tendon stands out, and the contraction keeps the foot flexed for a time which may be some minutes (in one case for twenty-seven); the relaxation is slow. Repetition lessens the contraction. The contraction is not voluntary (although a voluntary contraction may simulate it). It may occur on one side only. The contraction produced by brief faradism may be similarly prolonged in the same case. A similar contraction may be observed in the extensors of the toes, sometimes in the flexors of the knee, rarely in the arm muscles. Its mechanism is uncertain; whether reflex or not, whether produced through the muscle itself, or through the tension on the antagonists, (as Erlenmeyer has maintained), we do not know. It seems to be an excess of the contraction by which a muscle adapts itself to passive shortening of its course. In the cases that have presented this phenomenon there has been no muscular rigidity, and no great excess (even sometimes a loss) of the myotatic irritability. This adaptation to posture seems a phenomena quite separate from that which underlies myotatic irritability. It may occur in the early stage of tabes, and in some other affections, but its significance is not known. A phenomenon somewhat similar occurs in hysteria (Féré); it is indeed but an isolated and imperfect instance of the condition that when universal and perfect, constitutes the *flexibilitas cerea* of catalepsy. The nature of these contractions will be considered further on.

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[*] 'Diagnosis of Diseases of the Spinal Cord,' 2nd edit., 1881, p. 29.

[†] By some interesting researches recently published, Mommsen has reached the same conclusion, that muscular tone is dependent on a muscle-reflex action excited by tension acting on the sensory muscle-nerves ('Virchow's Archiv,' Bd. 110, p. 22).

[‡] 'Arch. f. Psych.,' Bd. x, p. 243.

CHANGES IN NUTRITION.—The nutrition of the tissue-elements is largely under the influence of the nervous system. Whether this influence is exerted through special "trophic" nerves, or through the motor, the sensory, and especially through the vasomotor nerves, is a question that has been much discussed and is still undecided. The balance of evidence is against the existence of special trophic nerves. The fact regarding these changes that is surest and most important, is that acute disturbance of nutrition is the result of irritation of the nerve-trunks and centres, and is in proportion to the intensity of that irritation. If a thread is passed through each sciatic nerve, and one is also irritated by the application from time to time of irritant liquids, trophic changes occur in that limb with greater rapidity, and far greater intensity, than in the other (Lewaschew).

The same fact is frequently indicated by the extreme readiness with which trophic lesions of the skin and even cellular tissue, occur in certain forms of inflammation of the spinal cord especially in those that have a strong tendency to spread in the cord, and hence may be regarded as possessing more of this peculiar quality that, in our ignorance, we term *irritative*. One theory and one only seems to explain all the facts. It is that the nerve-endings have a structural continuity with the tissues,—are related to them in a sort of molecular contact similar to that which exists in the tissues themselves—and that the nutrition of the molecules of the tissues is determined in its character by that of the nerves. The process of irritation descends the nerves, it passes from them to the tissues and invades them all. Thus we can understand equally the acute changes in the muscles which will be described, and also those in the skin and other tissues which are under the influence of the sensory fibres.

Nutritive changes in the skin are easily recognised, but they differ much in their character according to their acuteness. When they are rapid, and due to very acute irritation of the nerves or the spinal cord, such as those just mentioned, the temperature of the limb is raised, the vessels readily dilate, and remain dilated for a long time, bullæ form, containing a dark-coloured liquid, and slight pressure occasions a slough. These changes sometimes occur spontaneously, but are more often excited by some cutaneous irritation, and very slight irritation of the skin will suffice to produce them. Trifling pressure will set up a slough, and extensive vesication may result from the application of a hot-water bottle that is not more than pleasantly warm to a healthy hand. When the nerve irritation is intense, effusion may occur into the joints.

In the case of the fifth nerve, acute trophic changes occur in the eyeball chiefly when the disease involves the Gasserian ganglion, or the nerve in front of it. To produce the same effect, a lesion behind the ganglion has to be more irritating than one in front of it, and it is probable that a similar relation obtains in the case of the spinal nerves and the spinal ganglia. Very intense changes in nutrition are, however, common in acute inflammation of the spinal cord.

The alterations in nutrition in chronic lesions (which have been carefully studied by Paget and Weir Mitchell) differ considerably from those that result from acute irritation. There is a slow change in the nutrition of the skin, which becomes red, thin, and shiny—the "glossy skin" of Paget. The subcutaneous tissue also wastes, so that the finger tips become pointed. The growth of the hair and nails is retarded, and the latter become brittle. The bones may suffer in their nutrition, and may break more easily than in health. If the lesion occurs during the period of growth, this is retarded. These changes seem to depend on an altered life tendency of tissue elements that have been formed without some influence received from the nerves.

The *muscles* suffer from lesions of the motor nerves; the minute changes are described at p. 53. At first there may be merely flabbiness, and an appearance of wasting that is not confirmed by measurement, but there is soon an actual diminution in the circumference of the limb. In extreme cases all the muscular tissue disappears, and the contour of the limb is considerably changed. In comparing the size of the limbs on the two sides, it should be remembered that the limbs on the right side are normally somewhat larger than those on the left side, and that the difference varies according to the occupation of the individual, and the extent to which this involves an excessive use of the limbs on one side. In measuring, great care is necessary to secure, as nearly as possible, the same conditions on each side, both as regards the state of the muscles and the place of measurement. It is best, wherever possible, to take the maximum measurement in each part, rather than to attempt to make the measurement in the same place. In the calf, the maximum circumference should always be taken. In the thigh. accurate measurement is extremely difficult, because a maximum cannot be taken. We may endeavour to measure at the same point in the thigh, at the same distance from the condyle, but it is very difficult to be exact, and a more accurate comparison can often be made by taking the minimum circumference above the knee, in spite of the fact that the muscular tissue there is small, and the difference less than it is elsewhere. In the forearm the best result is obtained by taking the maximum measurement around the muscular prominence below the elbow, over the supinator longus. If the upper arm, the circumference is nearly the same in the middle third, and the middle of the humerus may be measured, with little risk of error. In all cases care must be taken to draw the tape equally tight at each place. It is easier to do this with a flexible steel measure than with an ordinary tape measure. Exactness in this respect is best secured by having a spring at one extremity of the tape, with an index that will show the tension, which can thus be kept the same at each place.*

Electrical Irritability.—The nerves and muscles are excitable by electricity, and the excitability is changed by disease, of which the change is often an important symptom. It indicates the state of nutrition of the nerve-fibres and muscles, and from this we can often draw important inferences regarding the condition of the centres. An outline of the most important facts regarding electricity in its application to medicine, is given in the Appendix, but it may be well to mention here the chief facts of diagnostic importance.

In the normal state, nerve-fibres are stimulated by either the induced or the voltaic current, the stimulation of the motor nerves being shown by contraction in the muscles supplied by them, that of the sensory nerves by the sensation that is caused. The contraction of the muscles is continuous when the faradic current is applied, but if the isolated shocks of which the current consists are separately passed, each causes a brief, momentary contraction. When the voltaic current is applied, contraction occurs, with a current of moderate strength, only when the strength of the current is changed, and chiefly when the current commences or ceases to pass, *i. e.* when the circuit is "made" or "broken." The stimulation of the sensory nerves is greatest at those times, but occur also, in a much slighter degree, during the whole time that the voltaic current is passing. Hence this is probably the case also in the motor nerves, although the stimulation is too feeble to produce a contraction in health with a strength of current that can be borne. In proportion as the nutrition of the nerve-fibres is impaired, their excitability is lowered and a stronger current of each kind is required to excite them and cause contraction in the muscles they supply. When their nutrition is much impaired—*i. e.* when the fibres are "degenerated"—no contraction can be obtained even with the strongest currents.

The changes in the excitability of the muscles are less simple, because in them there are two excitable structures—the terminations of the nerves, and the muscular fibres themselves. Of these the nerve-fibres are the more sensitive to faradism, and the faradic stimulation of a muscle under normal circumstances is by means of these motor nerve-endings. Thus we find that its excitability corresponds in degree to that of the motor nerve supplying it. The muscular fibres themselves are, even in the normal state, less sensitive to faradism than the nerve, apparently because they are incapable of ready response to a stimulus so very short in duration as are the shocks of which the faradic current consists. (The proof of this consists in the fact that under the influence of curara, which removes the excitability of the terminations of the motor nerve, the muscle requires a stronger faradic current to stimulate it than in the normal state.) But when the nerve is degenerated the slowly interrupted voltaic current stimulates the muscle as readily as in the normal state; a contraction occurs when the circuit is completed or broken—distinctly slower than that which occurs when the nerve-fibres are intact, and due to the stimulation of the protoplasm of the muscular fibres themselves. The fact that, under normal circumstances, the contraction which is caused by the voltaic current is as quick as that produced by the faradic shock, is ground for believing that, in health, the voltaic, as well as the faradic current, causes the muscle to contract chiefly by exciting the motor nerve-endings When the motor nerve is degenerated, and will not respond to faradic or voltaic stimulation, the muscle also loses all its power of response to the former. Apparently the nerve-degeneration is accompanied by changes in the nutrition of the muscular fibre, by which any power of response to faradism, which it possessed in the normal state, is lost. But the response to the voltaic current remains, and becomes quickly more ready than in health, doubtless in consequence of nutritive changes which develope what the older pathologists called, truly enough, "irritable weakness." Moreover, there may commonly be observed a change in the readiness of response to a certain mode of stimulation with voltaism—a "qualitative" change, as it is termed. In health, the first contraction to occur, on gradually increasing the strength of the current, is at the negative pole when the circuit is closed, and a stronger current is required before closure-contraction occurs at the positive pole. But, in the morbid state we are discussing, closure-contraction may occur at the positive pole as readily as at the negative, or even more readily—and contractions, when the circuit is broken, occur far more readily than in the normal state. This condition, then—faradic irritability lost, voltaic irritability increased and often changed in quality—is termed the "degenerative reaction," because it occurs when the nerve-fibres are degenerated; and if we test *them* we shall find no response to any stimulus, voltaic or faradic. It occurs when the nerves are separated partially or completely from their motor nerve-cells, and if no such separation exists, it indicates an acute degenerative change in those nerve-cells.

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* As in an instrument contrived by Dr. Russell Reynolds (and made by Hawksley) in which two rods, to be placed along the middle, one of the thigh, the other of the leg, are joined by a hinge that can be tightened at any angle, and on the upper one is a tape with the contrivance mentioned in the text. An analogous and most convenient tape measure.

But the motor nerve-cells and fibres often undergo changes in nutrition of a much more chronic character. In this condition the irritability of the fibres is lessened gradually and slowly. The irritability of the intramuscular nerve-endings is lessened in the same degree as that of the nerve-trunks, and we have a diminution to both faradism and voltaism. The nutrition of the muscular fibres is slowly, gradually, impaired; and when the nerve-fibres are much affected the muscular fibres are also. There is no stage in which the nerve-fibre irritability is lost, and the muscle-fibre irritability retained; hence there is no condition of lost faradic and increased voltaic irritability such as characterises the degenerative reaction just described. Irritability is changed to the one form of stimulus just as to the other.

Between these two forms there are intermediate conditions. For instance, the nerves may present normal irritability, and the muscle the increased voltaic irritability and changed order of contraction met with in degeneration. Probably, in these cases, some nerve-fibres are degenerated, and lead to the increased irritability of some muscular fibres. In both nerve and muscle the character of the reaction is determined by the more irritable structures; hence it is normal in the nerve and altered in the muscle. This has been termed by Erb the "middle form" of degenerative reaction. It would be more accurate to call it the "mixed form."

The various changes in irritability have been thought to indicate the existence, and various affection, of separate centres for the nutrition of the nerves and muscles, apart from, though acting through, the motor nerve-cells. Remembering that the nerves and muscles contain fibres which suffer in different degrees, the phenomena at present ascertained may all be explained on the simpler principle stated, without the assumption of these special centres, of the existence of which there is, indeed, no evidence.

Frequently the lowered irritability of degeneration in the nerves is preceded by a slight increase of irritability, very transient when the degeneration is acute, of longer duration when the degeneration is of the slower variety just noticed. In some morbid states, again, in which the change of nutrition in the cells and fibres is extremely slight, an increase may alone be discovered. I have found such an increase, for instance, in diseases regarded as functional, as paralysis agitans and chorea, and it is an interesting proof of the molecular changes which underlie, or result from, "functional" maladies.

THE MUSCLES: THEIR ACTION AND PARALYSIS.

Disease of the motor nervous system is largely manifested by loss of muscular action. Individual muscles, as well as groups of muscles, are often separately affected. Hence it is desirable to consider the symptoms of the paralysis of the more important muscles, before we enter on the study of special diseases.

The symptoms of the palsy of any muscle is a loss of its normal action, and a knowledge of this action is essential for the comprehension of those symptoms. The two must therefore be considered together. They are positive and negative aspects of the same facts.

It may be well, at the same time, to mention the nerve by which each muscle is supplied.

The action of muscles is threefold. (1) By their tonic contraction they maintain the parts in a certain posture, independently of voluntary effort. By actual contraction they (2) produce certain movements, and also (3) oppose the action of other muscles by a feebler contraction, and thus steady the movement that results.

The complex way in which muscles act together, and modify each other's effect, renders the subject a very large one. Here, only an outline can be given of the more salient facts concerning the most important muscles. The reader who desires to pursue the subject further can do so in the 'Physiologie des Mouvements,' of Duchenne, to whose researches our present knowledge is largely due.

The DIAPHRAGM (phrenic nerves from the fourth and fifth cervical) although a double muscle with two nerves, habitually acts as a whole, the two halves contracting simultaneously and diminishing each lateral curve of the arch. The central tendon descends but little. The abdominal viscera are depressed, and the parietes protruded. If the hand is placed beneath the ribs, the descent of the viscera beneath the diaphragm can be felt. When the diaphragm contracts alone, as when the intercostals are paralysed, or the phrenic nerve is faradised, the ribs to which the muscle is attached are slightly raised during its action, and this elevation causes a slight expansion of the thorax. In ordinary breathing this expansion is lost in the action of the intercostals. In paralysis the inspiratory protrusion of the upper part of the abdomen is lost; it even recedes during inspiration instead of advancing, and a descent of the viscera can no longer be felt by the hand. There often results a remarkable alternation in the respiratory movements of the thorax and abdomen, the retraction of the one corresponding to the protrusion of the other.

STERNO-MASTOID (spinal accessory nerve), passing from the sternum and adjacent part of the clavicle to the mastoid process, inclines the head towards, and rotates the face from, the side on which the muscle contracts. Both muscles together support the head in the vertical position, and, if it is bent back, they bring it forwards into, but not beyond, this position. Paralysis of one muscle has no influence on the position of the head, and but little on its movements. Other muscles supplement the loss. This is no such thing as a "paralytic torticollis." In palsy of both muscles, the head can be balanced in the vertical position, but if it falls back, it can be brought forward only with great difficulty. Each sterno-mastoid is associated in action with the muscles of the other side; it is a "contra-lateral muscle." For instance, in using the right arm, the head is turned to the right by the left sterno-mastoid. This association is sometimes reproduced in disease.

MUSCLES MOVING THE UPPER LIMBS.

MUSCLES MOVING THE SCAPULA AND SHOULDER-JOINT.—The *Trapezius* (special accessory, lower cervical and upper dorsal nerves) consists of three parts. The first, from the occipital bone to the outer end of the clavicle, is rarely used except in breathing (respiratory portion, Duchenne). The second part is that which passes from the lig. nuchæ, lowest cervical, and upper three dorsal spines, downwards and outwards to the acromion and outer part of the spine of the scapula. The lowest part passes from the dorsal spines below the third, outwards and partly upwards, to the inner half and base of the spine of the scapula. The

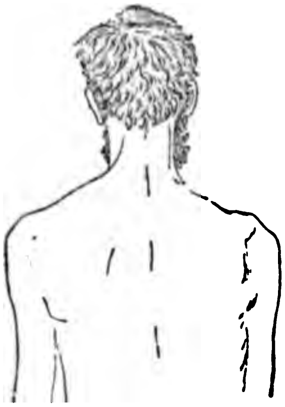

FIG. 5.—Paralysis and wasting of trapezius; alteration in contour of shoulder at rest.

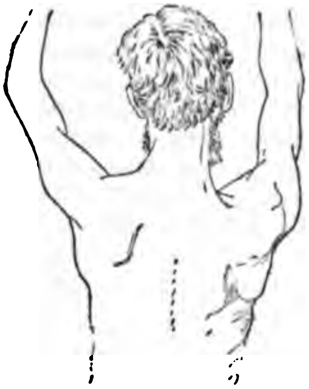

FIG. 6.—Ditto, when the arms are raised (the right one being aided by another person).

second part is the chief elevator of the scapula and shoulder. With the third part it brings the scapula towards the spine, and puts the shoulder back. Both parts tend to rotate the scapula—acromion up, lower angle out. By this rotation the arm is carried above the horizontal level, to which the deltoid raises it. Paralysis of the highest part has little influence on the movement of the scapula, but causes a change in the contour of the neck (Fig. 5) especially conspicuous on deep inspiration. The change in the shape of the neck is very great, when the arms are raised, if the whole trapezius is wasted (Fig. 6). In palsy of the middle part, the elevation of the shoulder is imperfect; in that of the third part, the scapula is farther from the spine than normal. In palsy of all parts, the scapula becomes rotated (acromion down, inferior angle in) by the weight of the arm and the contraction of the opponents (Fig. 7). The rotation may mask the displacement outwards, due to the paralysis of the lowest part. If the clavicular part remains, there may be no rotation, but the scapula is lower than normal.

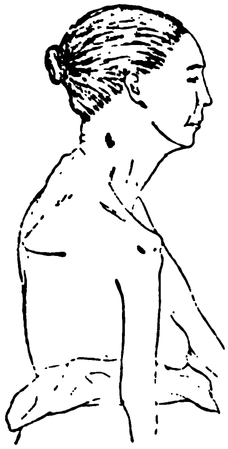

FIG. 7 —Paralysis and wasting of deltoid and trapezius; rotation of scapula, from weight of arm, in consequence of the paralysis of the trapezius; progressive muscular atrophy.

The *Rhomboids* (fifth cervical nerve, by a branch that passes through the scalenus) first rotate the scapula on the outer angle, moving the lower angle inwards, and then move the whole scapula upwards and inwards. In strong elevation, they aid the trapezius, which prevents the rotation of the scapula. The rotatory action aids forcible depression of the raised arm. The muscles also fix the scapula for the action of the teres major. Their tone helps to keep the scapula against the thorax (opposing the pectoralis) and in its vertical position (opposing the serratus), and hence, in paralysis, the edge of the scapula, at rest, stands out a little, leaving a furrow, and the scapula is slightly rotated (lower angle out). Movement is but little interfered with by the paralysis of the rhomboids, the most important effect being that the movement backwards of the raised arm by the teres and deltoid is feeble for want of the fixation of the scapula.

The *Levator anguli scapulæ* (third cervical nerve) first rotates the scapula on the outer angle and then raises it. The muscle is usually paralysed with the trapezius, and then the scapula falls, but the special effect of its palsy is lost in that of the trapezius. If the levator is preserved, and the trapezius paralysed, there is great rotation of the scapula, which is, as it were, suspended by its inner angle (Fig. 7).

Serratus magnus (posterior thoracic nerve, from the fifth and sixth cervical) carries the scapula outwards, forwards, and slightly upwards, when the arm is put forwards. It tends to rotate the scapula on the inner angle (acromion up), the lower fibres most powerfully, but this rotation is prevented by the rhomboids and levator anguli. It does not raise the shoulder when the arm is hanging. It helps to fix the scapula when the posterior fibres of the deltoid move the raised arm back. If the scapula is fixed by the rhomboids, the serratus can act on the ribs, and aid forced inspiration. It has most inspiratory effect when the arms are elevated. In paralysis there may be little change in the position of the scapula at rest, but often there is slight rotation (lower angle in) from the unopposed tone of the rhomboids. When the arm is moved forwards by the anterior part of the deltoid, the scapula, no longer held against the thorax and moved forwards by the serratus, is rotated on its vertical axis by the action of the anterior part of the deltoid on the humerus, and of the middle part on the scapula. Thus the posterior edge recedes from the thorax, leaving a groove into which the hand can sometimes be placed (Fig. 8). The scapula is, at the same time, rotated; lower angle inwards and upwards. Elevation of the arm above the level of the shoulder is much weakened, but can be imperfectly effected by the middle part of the trapezius. Loss of the serratus weakens other movements, but does not abolish any. Inspiratory expansion of the thorax, when the arms are raised, is distinctly less on the paralysed side (Poore).

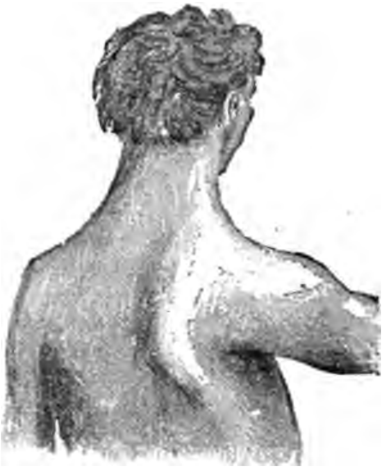

FIG. 8.—Paralysis of the serratus magnus; eversion and rotation of scapula when the arm is put forwards.

The *Deltoid* (circumflex nerve, from the four lowest cervical) abducts the humerus, the anterior and posterior fibres also moving the arm forwards and backwards respectively. The arm is raised least by the posterior, and most by the anterior fibres, but even the latter only elevate it to a right angle with the trunk. Hence, if raised by the anterior fibres, and then moved back by the posterior, it is at the same time depressed. Elevation above a right angle is by rotation of the scapula (trapezius and serratus). These muscles also fix the scapula for the deltoid, preventing the rotation (acromion down, lower angle in) that the deltoid, acting alone, would cause. In paralysis, abduction of the arm, direct, forwards, and backwards is almost lost. All the abduction that remains is a trifling movement by the supraspinatus. An attempt to abduct results in rotation of the scapula and elevation of the shoulder (Fig. 9) from an excessive innervation of the associated trapezius and serratus, which, as we have seen, fix the scapula when the deltoid acts. Paralysis of single parts of the deltoid causes loss of the corresponding movements of the arm, but if the middle part only is paralysed, there is still a limited power of direct abduction by the conjoined contraction of the anterior and posterior parts, aided by the supraspinatus.

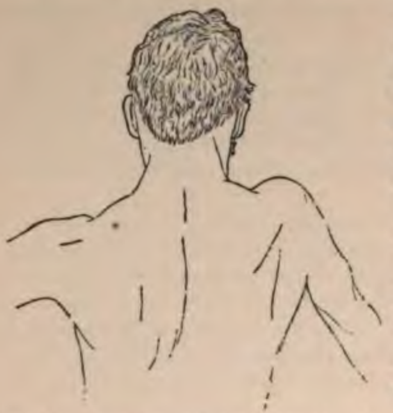

FIG. 9.—Paralysis of right deltoid; elevation of shoulder by trapezius on an attempt to raise the arm, which is slightly abducted by the supraspinatus.

The *Supraspinatus* (suprascapular nerve) abducts the arm, moves it forwards, and rotates it in. It thus aids the deltoid. Isolated paralysis of the supraspinatus has little influence on movement or position, but if the deltoid is also paralysed, the head of the humerus falls away from the acromion far more than when the deltoid is paralysed alone.

The *Infraspinatus* (suprascapular nerve) rotates the humerus outwards, and in paralysis this movement is lost. A difficulty in writing is produced, the movement along the line being by this rotation of the humerus.

The *Teres minor* (circumflex nerve) has a similar action to the infraspinatus, and its palsy has a similar effect.

The *Subscapularis* (short subscapular nerve, from the fifth and sixth cervical) rotates the humerus in, and its paralysis lessens this movement.

The *Latissimus dorsi* (long subscapular nerve, from the brachial plexus—last four cervical nerves) lowers the raised arm, and puts it back; the upper part adducts the scapula, the lower depresses the shoulder by acting on the humerus, which it tends to drag out of the socket. It inclines the trunk a little, and both muscles together extend the trunk. In paralysis, forcible backward depression of the raised arm is lost, and the shoulder cannot be put back without being also raised (by the trapezius).

The *Pectoralis major* (anterior thoracic nerves from the brachial plexus) consists of two muscles, the action of the clavicular and sternal parts being different, The clavicular (which arises also from the highest part of the sternum), if the arm is hanging, brings the shoulder forwards and upwards, as if shivering; if the arm is raised, it is brought forwards and lowered to the horizontal position. The muscle is thus concerned, Duchenne says, in the "cut" of the swordsman and the benediction of the priest. The sternal portion lowers the raised arm from every position, and if the arm is hanging, it draws the shoulder down. Paralysis of the upper part has little effect on the movement of the arm, because the anterior fibres of the deltoid have the same action. It is easily recognised by making the patient put his arms in front of him and press the palms together. In paralysis of the lower part, even with the lattissimus, the raised arm can still be lowered accurately, by the weight of the arm and relaxation of the elevators, but it cannot be lowered against even a slight resistance. Thus a blacksmith with this defect could wield a heavy hammer, but could not blow the bellows by pulling downwards a cord (Duchenne).

The *Teres major* (short subscapular nerve) approximates the humerus and the outer edge of the scapula, by bringing the former to the side of the trunk, and rotating the latter. The simultaneous contraction of the levator anguli and rhomboids, fixing the inner angle, causes this rotation to raise the prominence of the shoulder Hence, in a forcible elevation of the shoulder, the arm is pressed

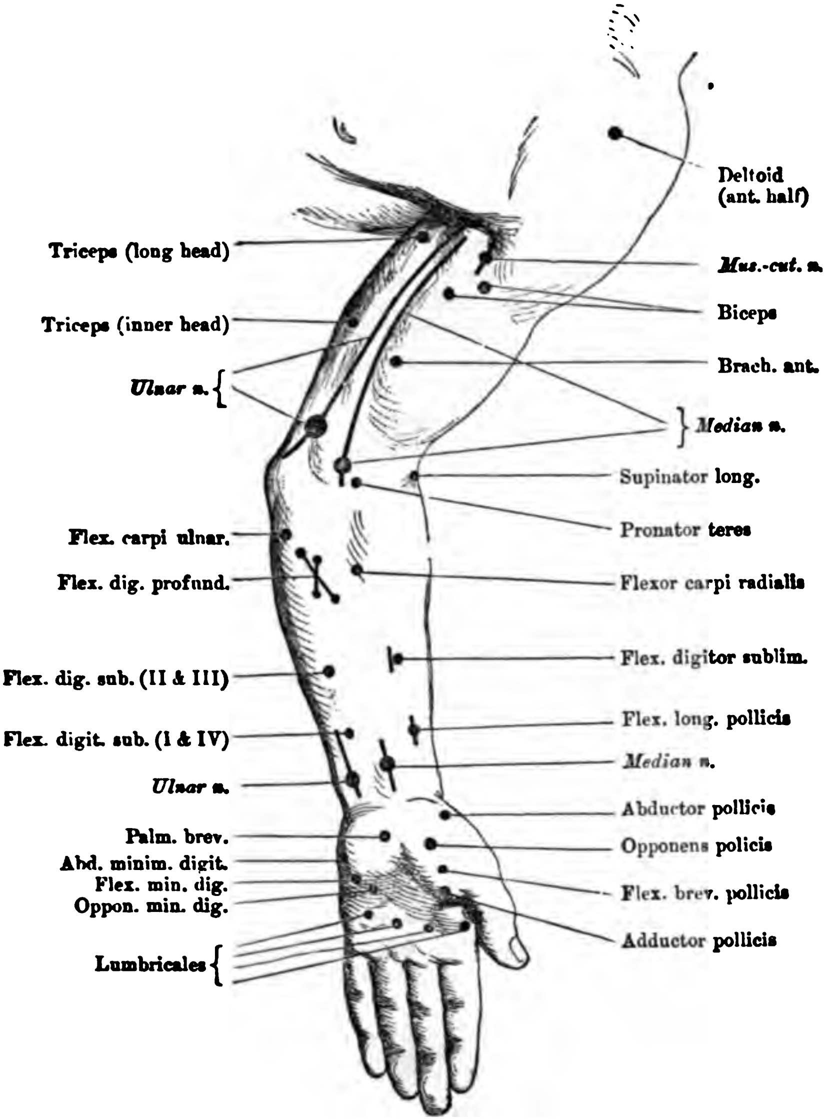

FIG. 10.—Motor points for the arm, inner side (from Erb). The points at which the muscles and nerves can most effectively be stimulated.

against the side, the lower fibres of the latissimus and pectoralis major aiding the adduction. The teres cannot alone put the arm behind the trunk. In paralysis, the elevation of the shoulder, with the arm against the side, is lost.

MUSCLES MOVING THE FOREARM.—*Triceps* (musculo-spiral nerve).—The long head has an action similar to the teres, but feebler. It contracts when the arm is forcibly lowered, and prevents the displacement downwards of the head of the humerus by the actual depressors, the latissimus and pectoralis. All parts extend the elbow, the long head with less force than the others, but its action on the shoulder-joint, just mentioned, is important, because forcible depression of the raised arm is often associated with extension of the elbow. In paralysis of the triceps, the elbow can only be extended by the weight of the forearm, and extension against gravitation is impossible. Thus a man with paralysis of the triceps cannot raise his hat in the customary manner. Flexion of the elbow is uncertain, on account of the loss of the antagonistic steadying force.

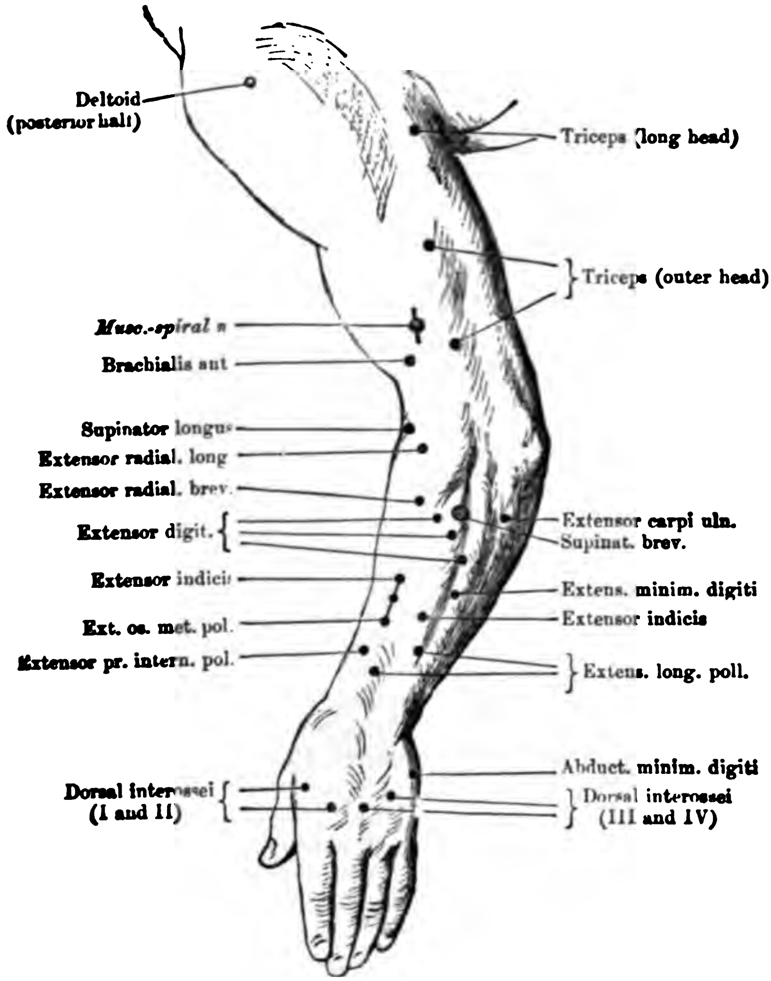

FIG. 11.—Motor points for the arm, outer side (Erb).

The *Brachialis anticus* (musculo-cutaneous and musculo-spiral nerves) flexes the elbow simply. Its rare isolated palsy has little effect, since it is supplemented by the biceps and supinator longus.

The *Biceps* (musculo-cutaneous nerve) supinates the forearm if it is pronated, and then flexes the elbow. In paralysis the flexion can still be effected, but the traction on the humerus causes pain at the shoulder, from the loss of the support of the long head of the biceps.

The *Supinator longus* (musculo-spiral nerve) places the forearm midway between pronation and supination, and then flexes the elbow. If it is paralysed, there is a tendency for supination to accompany flexion (biceps); and if the brachialis anticus is also paralysed, the elbow can only be flexed when the forearm is supinated.

If the three direct flexors of the elbow are paralysed, feeble flexion is still possible by the extensors of the wrist, which cross the elbow-joint, but only after the forearm has been pronated and the wrist over-extended.

The *Supinator brevis* (musculo-spiral nerve by posterior interosseal branch) is the only simple supinator. If it is paralysed, supination can still be effected by the biceps, and also by the supinator longus as far as midway between pronation and supination.

Pronators.—The *p. teres* and *p. quadratus* (median nerve) both pronate strongly, and their palsy causes loss of this movement, but pronation to the mid-position is still possible by the supinator longus.

MUSCLES MOVING THE HAND.—*Flexors of Wrist.*—The *F. carpi ulnaris* (ulnar nerve) and *F. carpi radialis* (median) flex the wrist. The ulnar flexor tends to turn the supinated hand still more out, but neither moves the wrist-joint laterally. Flexion indeed hinders the lateral movements of the wrist, in consequence of the shape of the articular surfaces. The f. c. ulnaris flexes the fifth metacarpal bone on the carpus, as well as the wrist-joint. In paralysis, flexion of the wrist can only be effected by the flexors of the fingers when these are extended. Extension of the wrist is unsteady from the loss of the antergic contraction of the flexors (see p. 7).

Extensors of Wrist.—Extensor carpi radiales brevis et longus; E. c. ulnaris (musculo-spiral nerve and its radial branch). The short radial is a direct extensor; the long radial and the ulnar move the hand laterally as well. In paralysis of all three extensors the wrist can only be extended by the extensors of the fingers when the phalanges are flexed. In loss of the short radial, direct extension is still possible by the long radial and ulnar, and lateral extension by one of these alone. If either is paralysed, together with the short radial, direct extension is lost, and only lateral extension, in the direction of the remaining muscle, is possible. Paralysis of either lateral extensor, long radial or ulnar, leads to permanent deviation of the wrist in the direction of the remaining muscle (Fig. 12). The loss of the long radial is more serious than that of the ulnar, because the radial lateral movement is of more importance, being needed for the convenient motion of the hand to the mouth. Paralysis of the extensors impairs flexion of the fingers, from the great shortening of the course of the tendons, by the flexion of the wrist that occurs. The ulnar extensor acts

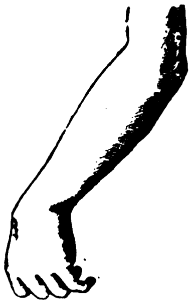

FIG. 12.—Paralysis of the long radial extensor of the wrist in a young child; habitual deviation of the hand towards the ulnar side. (After Duchenne.)

also synergically with the extensor of the metacarpal bone of the thumb, as may be noted if the finger is placed on the tendon beneath the styloid process of the ulna. Hence, in paralysis of this extensor, the hand deviates laterally when the thumb is strongly extended.

Extensors of the Fingers.—Extensor communis digitorum; E. indicis; E. minimi digiti (musculo-spiral nerve). The common extensor moves the fingers and then the wrist. When the muscle is faradised, the extension begins at the distal phalanges, and these become flexed again, when the hand is extended beyond the plane of the forearm, by the tonic force of the flexors, the course of their tendons being elongated by the extension of the wrist. Moreover, the muscle has little action on the last two phalanges, since they cannot be extended by the long extensor, if the interossei, their proper extensors, are paralysed. During extension by the communis the fingers are separated from the second. The extensors of the first and last fingers have a similar extensor action, but, in addition, they adduct their respective fingers towards the middle finger. In paralysis, the extension of the fingers is impossible, but if the proximal phalanges are passively extended, the middle and distal joints can be extended by the interossei. For the lateral movements of the digits, extension of the proximal phalanges is essential, and hence these movements are lost, but they can be performed if the proximal phalanges are passively extended. The posture of the fingers due to contraction of the palmar fascia resembles that in palsy of the long extensor (see Fig. 13), but an examination of the palm shows the cause of the flexion.

FIG. 13.

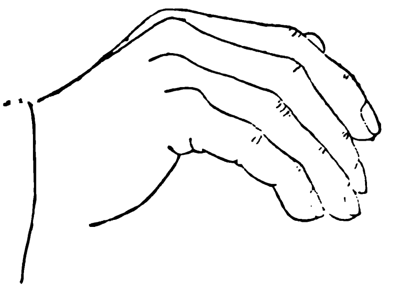

FIG. 14.

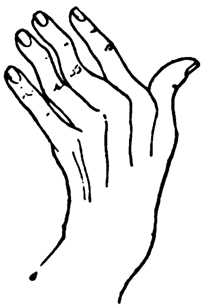

FIG. 13.—Posture of the hand in contraction of the palmar fascia, resembling that in paralysis of the long extensors of the finger.

FIG. 14.—Paralysis of the fibres of the flexor sublimis which act on the two middle fingers: twelve years' duration. The second phalanges of these fingers are bent backwards and subluxated from the contracture of the unopposed interossei, while the last phalanges are kept in position by the unaffected flexor profundus. (After Duchenne.)

Flexors of Fingers.—*F. sublimis* (median nerve); *F. profundus* (median and ulnar nerves). These muscles flex chiefly the second and third phalanges, the first phalanx being flexed by the interossei. The superficial muscle flexes the second phalanx on the first, the deep flexes both. The action on the first phalanx is confined to extreme flexion of the fingers, and is the less, the more the wrist is flexed. But if the flexion of the middle and distal phalanges is prevented the first is strongly flexed. **In extreme shortening of the course of the tendons** by flexion of the wrist, the action on the fingers is very feeble, evidence of a normal antergic action of the extensors of the wrist. When the extensor of the fingers is in strong action, extending the proximal phalanges, the action of the flexors on the second and third joints is very strong (tearing position). In paralysis of these muscles the power of flexing the last two joints is lost, but the interossei still flex the metacarpo-phalangeal joints. Paralysis of the deep flexor alone causes merely loss of the power of flexing the distal joint, but this impairs many movements, such as playing on the piano. In paralysis of these muscles, the unopposed tone of their opponents, the interossei, which extend these joints, leads in time to over-extension, and, with repeated passive pressure in using the fingers, may even produce a subluxation backwards. In palsy of the sublimis this effect is chiefly seen at the middle joint (Fig. 14), in that of the profundus at the distal joint.

Interossei and Lumbricales (ulnar nerve, except the outer two lumbricales, which are supplied by the median).—The Interossei abduct and adduct the fingers, but only when these are extended at the metacarpo-phalangeal joints, and some effort is required for adduction, since the tendency of the long extensor is to separate the fingers, and this influence has to be overcome. They also extend the second and third phalanges on the first, and flex the first on the metacarpal bones. The lumbricales aid the flexor-extensor action of the interossei, but do not move the fingers laterally. The opposite action of the forearm muscles and of the interosseal extensors and flexors is very important. Their synergic action steadies movements, and in many actions they contract alternately. Thus in making a down-stroke with a pen or pencil the long flexors bend the last two joints; while in making an upstroke these are extended, and the metacarpo-phalangeal joint is flexed, by the interossei. In paralysis of these muscles the lateral movements are lost, but a slight abduction and adduction of the index can still be effected by its long extensors. Only the first phalanx can be extended, and flexion is almost confined to the last two phalanges. The first three lumbricales, being supplied by the median nerve, often escape when the other muscles are paralysed by an injury to the ulnar nerve, and they aid the others when these are merely weak; hence the index and middle fingers seem to recover before the others (Fig. 15). The position of the hand at rest becomes altered. Normally there is slight flexion at all joints by the tone of the muscles, interossei and long flexors. In paralysis the first

FIG. 15.

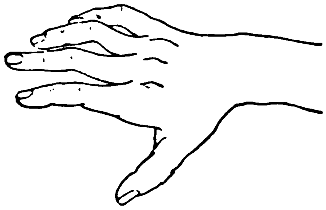

FIG. 16.

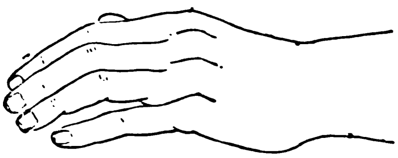

FIG. 15.—Recent incomplete paralysis of the interossei from a punctured wound of the ulnar nerve at the wrist: attempt to extend fingers. The loss of extension of the last two phalanges is chiefly marked in the third and fourth fingers, from the influence of the lumbricales (supplied by the median) on the others. (After Duchenne.)

FIG. 16.—Paralysis of the interossei (ulnar nerve) slight in degree: attitude of fingers at rest.

phalanx is in a line with the metacarpal bones, while the other phalanges are flexed, the middle more than the distal (Fig. 16). In action this flexion is always increased, the metacarpo-phalangeal joints become over-extended, and the other joints strongly flexed (Fig. 17). Gradually the hand assumes this posture even at rest (Fig. 18), and ultimately the posture becomes warped into a deformity by the over-extension of the first phalanges, and extreme flexion of the others, due to the contracture of the long extensor and of the flexors; the tendons of these muscles stand out conspicuously on the back and in the palm, and a claw-like attitude is developed, the "main en griffe" (Figs. 19 and 20). Changes in the articulations may ultimately limit even passive movement.

FIG. 17.

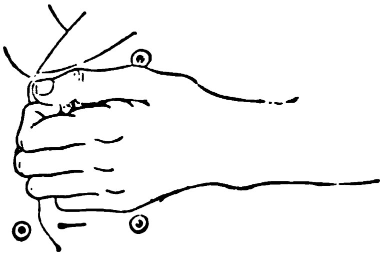

FIG. 18.

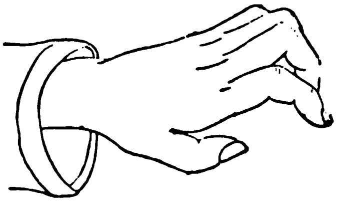

FIG. 19.

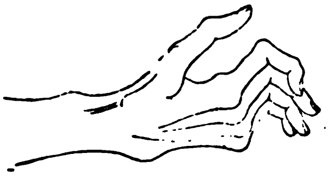

FIG. 20.

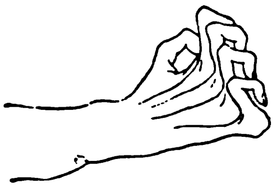

FIG. 17.—Attempt to unbutton waistcoat by the hand shown in the last figure; extreme flexion of the last two phalanges, and extension of the first, on the attempt to use the fingers. (From nature.)

FIG. 18.—Old-standing palsy of interossei and thenar muscles, showing the over-extension of the first and flexion of the last two phalanges.

FIG. 19.—Paralysis of all the intrinsic muscles of the hand and of the long flexor of the thumb, in consequence of an injury to the brachial plexus in dislocation of the shoulder. The fingers present the claw-like attitude; the thumb is in extension. (After Duchenne.)

FIG. 20.—Paralysis of the ulnar nerve from a wound at the wrist (indicated in the figure). Extreme claw-like hand from the unopposed contraction of the common extensor and long flexors of the fingers and thumb. (After Duchenne.)

MUSCLES OF THE THUMB.—*Extensor secundi internodii pollicis* (musculo-spiral nerve) extends both phalanges, and moves the whole thumb backwards and from the fingers, so as to bring it behind the plane of the metacarpus. It may ultimately extend the wrist-joint, but it never supinates. It is not used in extending the thumb when this is opposed to the first finger. In paralysis, the metacarpal bone of the thumb is slightly flexed on the carpus, and is inclined forwards. The second phalanx is flexed on the first, and can only be extended (by the abductor and outer part of the short flexor) when the metacarpal bone is adducted and the first phalanx is flexed. The constant flexion of the second phalanx interferes with the movement of the index finger, unless the patient remembers to move the thumb out of the way by the extensor of the metacarpal bone. Writing is not interfered with because the muscle is not concerned in extension with opposition.

The *Extensor primi internodii pollicis* (musculo-spiral nerve) is the true abductor of the thumb. It moves the metacarpal bone outwards, and extends the first phalanx. It would move the whole hand in the same direction as the thumb, were not this tendency counteracted by the antergic contraction of the extensor carpi ulnaris (q. v.). It does not pronate or supinate. In paralysis abduction of the metacarpal bone is less than normal. There is an undue flexion of the first phalanx, and the metacarpal bone is flexed on the carpus, so that the thumb is drawn towards the palm. The loss of this muscle is, however, compensated to a considerable extent by other muscles.

The *Extensor ossis metarcarpi pollicis* (musculo-spiral nerve) is really the long abductor of the thumb. It moves the metacarpal bone outwards and forwards, flexing it on the carpus, and then flexes the wrist with slight pronation. It thus moves the thumb as much forwards as outwards. In *paralysis* the metacarpal bone is, at rest, less inclined forwards than normal, and somewhat abducted, but the first phalanx is in its normal position. Movement of the thumb is but little interfered with.

In combined palsy of the extensors of the first phalanx and of the metacarpal bone the thumb becomes adducted, and is parallel to the radius. The first phalanx is slightly flexed by the thenar muscles.

The *Thenar muscles* constitute two groups: (1) The short *abductor and outer portion of the short flexor* (median nerve) move the metacarpal bone forwards and inwards (flexing the first phalanx), incline it outwards, and rotate it inwards, so as to place its palmar aspect opposite the fingers. The second phalanx is ultimately extended. If the metacarpal bone is previously abducted the movement is greater, and amounts to circumduction. (2) The *adductor and inner part of short flexor* (ulnar nerve) go to the inner side of the first phalanx. The metacarpal bone is moved towards that of the second finger: if previously flexed, it is extended; if previously opposed to the index, it is moved a little outwards. The phalanges follow the movements of the metacarpal bone, but the first is slightly flexed and the second is extended, as the fingers are by the interossei.

The *Opponens pollicis* (median nerve) flexes the metacarpal bone on the carpus, and abducts it, but this movement is insufficient to oppose the thumb to the index; the conjoint action of the abductor is necessary (see above).

The *Flexor longus pollicis* (median nerve) flexes the second phalanx forcibly and the first feebly. It has no action on the metacarpal bone. It is used in writing (making a stroke towards the body) and in picking up a small object, &c. In paralysis this flexion is lost, and with it these actions; if an object is held between the tips of the thumb and forefinger, the last phalanx of the thumb is bent back. Other movements of the thumb are not interfered with.

If all the thenar muscles are paralysed the metacarpal bone is in the plane of the index, and drawn towards it by the extensor of the second phalanx, which moves the metacarpal bone inwards and backwards. The whole thumb corresponds with the metacarpal bone in position, the phalanges being normal. In paralysis of the short abductor and flexor the second phalanx cannot be extended unless the metacarpal bone is abducted. These short muscles normally prevent the abduction that the extensor of the second phalanx tends to produce. The latter, moreover, prevents undue adduction when the special abductors are paralysed, but brings the metacarpal bone into the plane of the index. Thus the posture of the hand at rest resembles that of the hand of an ape (Figs. 21 and 22). If the abductor and opponeus are paralysed, the tips of the thumb and fingers can only be brought together by flexing the last phalanges of the digits (Fig. 24). Then the thumb can be brought into contact with the finger by means of the short flexor, which inclines the metacarpal bone sufficiently to effect this, although not enough for the tip of the first finger to touch the thumb when its phalanges are extended.

FIG. 21.

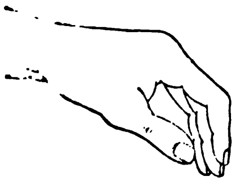

FIG. 22.

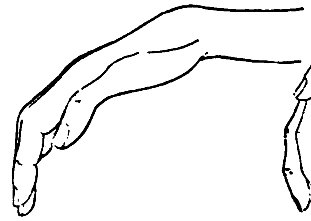

FIG. 23.

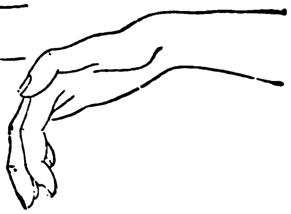

FIG. 21.—Normal position of the thumb (for comparison with the succeeding figures).

FIG. 22.—Position of the hand in long-standing paralysis and wasting of the thenar muscles. Under the influence of the long extensor the metacarpal bone of the thumb has been brought into the same position as the other metacarpal bones, being rotated slightly, so that the back of the thumb is in the plane of the back of the hand, like the hand of the ape. (After Duchenne.)

FIG. 23, from another case, shows the same condition, but still greater displacement of the metacarpal bone has taken place, from the greater contraction of the extensor. (After Duchenne.)

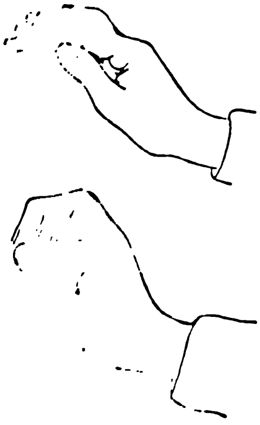

FIG. 24. Paralysis of the abductor brevis and opponens pollicis. From the want of these muscles the thumb can only be brought in contact with the tip of the index by strong flexion of the last two phalanges of the fingers, otherwise the tip of the thumb only reaches the middle of the second phalanx. (After Duchenne.)

If the short flexor is paralysed, the thumb can still be opposed to the first two fingers by the abductor, but it cannot be opposed to the last two fingers, on account of the deficient lateral inclination of the thumb, which should be produced by this muscle. Writing is easy by means of the short abductor, whereas, if this is lost, although the thumb can be opposed to each of the fingers, writing is much interfered with.

If all the thenar muscles are paralysed, a certain amount of opposition of the thumb and fingers is still possible, by means of the flexion of the last phalanges of the thumb and fingers. If all are paralysed except the adductor, objects can still be held between the thumb and side of the palm.

MUSCLES OF THE LOWER LIMB.

MUSCLES MOVING THE HIP-JOINT.—The *Gluteus maximus* (small sciatic nerve chiefly) extends the hip-joint, and freely rotates the thigh outwards. It is the most powerful extensor of the hip, and it is chiefly used when a forcible extension is required, and the joint has been previously flexed. It is employed, not in standing, or in walking on level ground, but in going upstairs, or uphill, and in rising from a seat. When it is paralysed these movements are difficult.

The *Gluteus medius* (gluteal nerve) is the chief abductor. All parts of the muscle have this action, but, in addition, the anterior third moves the thigh forwards and rotates it inwards, while the posterior third moves it backwards and rotates outwards. The successive action of the several parts causes circumduction. The *Gluteus minimus* (gluteal nerve) has probably the same action. In paralysis, abduction and circumduction are lost; in standing on the other foot the pelvis is inclined from the affected side, and hence, in walking, there is an oscillation of the trunk, which becomes very considerable if the muscles of both sides are affected. Moreover, the unopposed tone of the outward rotators produces a permanent rotation of the leg, so that the toes are directed outwards, and, from the altered position of the foot, the propulsion of the body in walking is deficient.

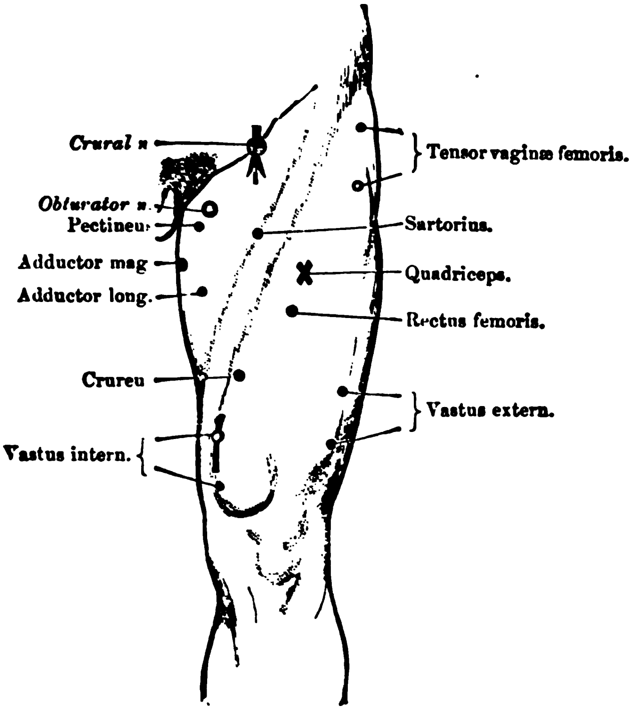

FIG. 25.—Motor points on the front of the thigh (Erb).

The *Pyriformis*, *Gemelli*, *Obturator internus*, and *Quadratus femoris* (special nerves from the sacral plexus) all rotate the thigh outwards, and the first-named muscle, in addition, carries the thigh obliquely backwards and outwards, in the same way as the posterior fibres of the gluteus medius. In paralysis of these muscles external rotation is impossible, and the unopposed tone of the internal rotators (anterior fibres of the gluteus medius and minimus) causes the leg and foot to be habitually turned inwards.

The *Psoas* (lumbar nerve) and *Iliacus* (anterior crural nerve) flex the hip-joint, and, in doing so, cause also a slight rotation outwards. In paralysis, flexion is lost, and the use of the leg in walking becomes impossible.

The *Tensor vaginæ femoris* has a slight power of flexing the hip, and at the same time rotates the thigh in. It normally counteracts the tendency of the ilio-psoas to rotate outwards. If it is paralysed, there is a tendency for the foot to turn out when it is being brought forward in the act of walking.

Adductors of the Thigh.—The *Pectineus* (obturator nerve) causes an oblique movement forwards and inwards, *i. e.* a combined flexion and adduction, as in crossing the legs. It also rotates outwards. The *Adductor longus*, and probably the *Adductor brevis* (obturator nerve), have the same action, but the flexion is less than by the pectineus. The *Adductor magnus* (obturator and great sciatic) causes a similar adduction, but while its upper fibres rotate outwards its lower fibres rotate in, and are employed in keeping the foot straight during adduction in riding. This is very difficult if these fibres are paralysed. The foot then turns out when the hip is flexed, either in the recumbent posture or in walking, from the preponderance of rotation out by the other adductors. When all the adductors are paralysed, not only is adduction lost, but in flexion of the hip the foot is moved forwards and outwards, instead of directly forwards, showing that there is normally a synergic action of the abductors and adductors with the flexors in this movement.

MUSCLES MOVING THE KNEE.—*Extensors: Rectus, Vasti,* and *Crureus,* together called the *E. quadriceps* (ant. crural nerve).—The vasti act solely on the knee-joint; the rectus also aids in flexing the hip, but chiefly when the knee is bent. In consequence of its passage over the hip-joint, moreover, the force with which it extends the knee is increased by the simultaneous extension of the hip. This effect is useful in the propulsion forwards of the body in walking. The crureus is unimportant.

In paralysis of the extensors of the knee, standing is still possible if the knee is extended, since the arrangement of the articulation renders a contraction of the extensors unnecessary. But secondary shortening of the flexors is apt to occur, and then standing becomes impossible because the knee cannot be perfectly extended. In the same way, walking is possible if the leg is not moved forward beyond the vertical position; if it is, the knee becomes flexed by the weight of the leg and foot, and the patient falls when he attempts to rest upon it. Rising from the kneeling posture in the ordinary way is impossible. In partial paralysis of the muscles, as in pseudo-hypertrophic paralysis (q. v.), the extension of the knee, in rising, is facilitated by placing the hand upon it, and so bringing the centre of gravity of the body near the fulcrum of the lever formed by the femur. If the vastus internus and rectus are paralysed, the vastus externus may dislocate the patella by the obliquity of its traction. The vastus internus never does so in the opposite condition, because its action is less oblique.

Flexors of the Knee.—The *Sartorius* (ant. crural nerve) flexes the hip- and knee-joints, and has a feeble power of rotating the thigh outwards and the knee inwards. It is a muscle of small importance.

The *Gracilis* (obturator nerve) adducts the thigh more powerfully than it flexes the knee. It rotates the leg inwards.

The *Semitendinosus, Biceps,* and *Semimembranosus* (great sciatic nerve) are not only flexors of the knee but extensors of the hip-joint, and are the muscles that extend the hip during ordinary walking, the gluteus maximus (q. v.) being called into action only during special efforts. The leg is rotated inwards by the semitendinosus, outwards by the biceps.

In paralysis of the flexors, the resulting loss of the power of flexion interferes with walking, since the knee-joint cannot be bent, in the forward movement of the leg, until the thigh is flexed sufficiently to permit the weight of the foot to flex the knee. To prevent the toes striking the ground the foot is unduly flexed on the leg. The loss of the support that the flexor tendons give to the knee-joint leads to an undue strain on the ligaments, which become stretched, and slight retroflexion of the joint may occur.

In paralysis of the muscles that extend the hip, there is a tendency to fall forwards in walking. To counteract this the trunk is carried backwards, and a fatiguing strain on the flexors of the hip results.

In paralysis of the biceps, the leg, during flexion, is rotated inwards; when the biceps remains and the other muscles are paralysed, there is an undue rotation outwards. The effect of these abnormal movements on the ligaments of the joint is such that, after a time, the amount of rotation becomes greater than is possible in health.

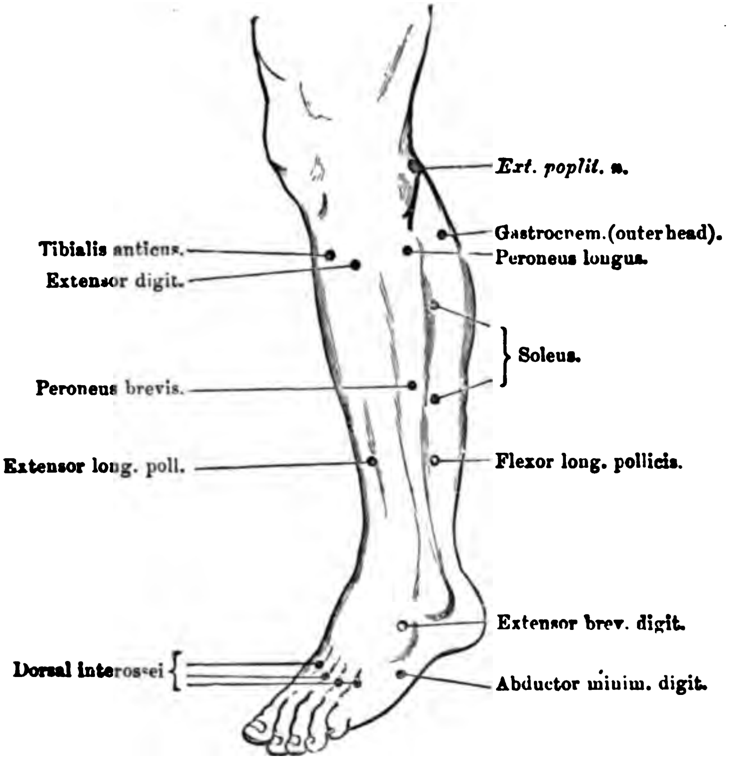

FIG. 26.—Motor points of leg, outer side (Erb).

The *Popliteus* (internal popliteal nerve) has but a feeble power of flexing the knee. Its chief action is to rotate the leg inwards when the knee-joint has been flexed.

MUSCLES MOVING THE FOOT.—*Extensors of Foot on Leg.**—The *Gastrocnemius*

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* These muscles are sometimes termed "plantar flexors," because they are homologous with the flexors of the wrist. The term is a bad one, since it involves a use

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and *Soleus* (internal popliteal nerve, from the sciatic) have the same action. They extend the hinder part of the foot and draw down the outer side of the forepart of the foot, but very little the inner side. Hence the foot is rotated, so that the dorsum looks outwards, while the whole foot is turned inwards on the axis of the leg. The peculiar inversion and adduction that thus accompanies

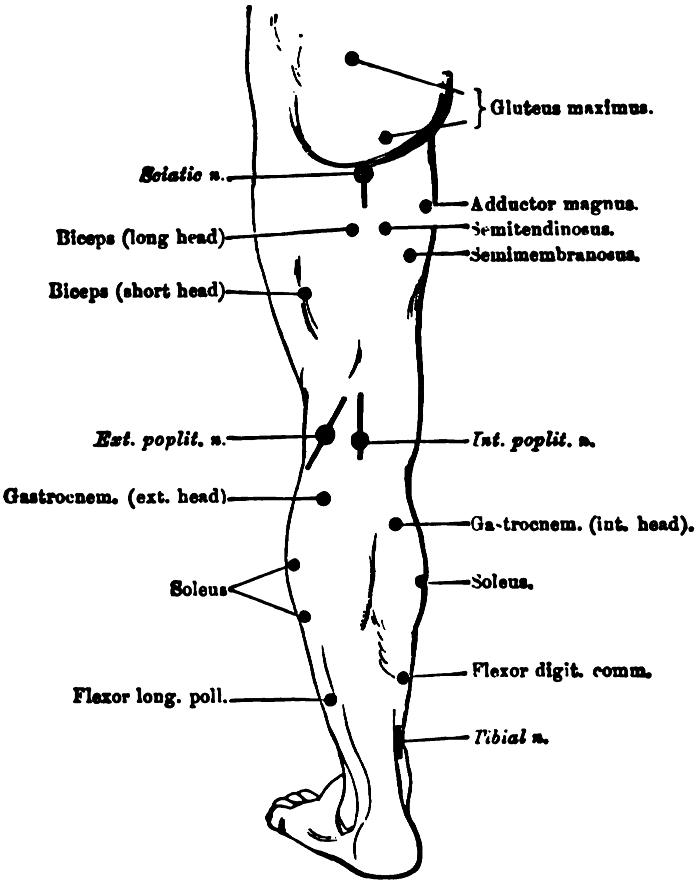

FIG. 27.—Motor points, back of thigh and leg (Erb).

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of the word "flexor" in absolute contradiction to its proper signification. We ought not to frame a descriptive term such as this on an analogy which involves a contradiction to the description. Flexion is bending, a movement from a straight line; and extension is less bending, a movement towards a straight (stretched, extended) line. To call a movement toward a straight line "flexion," because in the arm the similar movement produces this effect, is a process that is not description, but the statement of an analogy—the worst possible kind of nomenclature. Names should be descriptive or they should be arbitrary. Neither theory, nor analogy, nor homology should have any part in them. What we call homology is merely a species of analogy; it rests on inference and reasoning, not on simple absolute aspect. Nothing that rests on reasoning is inherently stable and free from change.

extension is due to the form of the articular surfaces. The gastrocnemius has very little power of flexing the knee, but the extension of the knee increases the effect of the muscle on the ankle-joint, especially in walking, just as we have seen that the extension of the hip augments the force with which the contracting rectus extends the knee. For direct extension of the ankle, the peroneus concurs and opposes the inversion. The only difference between the gastrocnemius and soleus is that the latter, having no attachment to the femur, can extend the ankle when the knee is flexed as well as when it is extended. In paralysis of these muscles, extension of the ankle (by the peroneus longus and flexor longus digitorum) is extremely feeble, and the foot can scarcely be carried beyond a right angle. Walking is greatly interfered with; standing on tiptoe is impossible. The unopposed peroneus longus causes eversion of the foot, lowers the head of the first metatarsal bone, and deepens the plantar arch. In time the ankle-joint becomes over-flexed, the heel considerably lowered, and the plantar muscles and fascia become shortened. The resulting deformity is termed talipes calcaneus (Fig. 28).

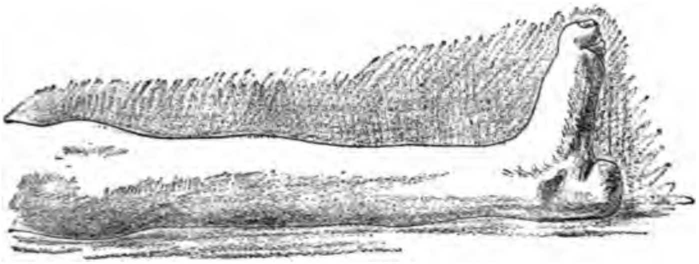

FIG. 28.—Talipes calcaneus from atrophic paralysis of the calf muscles, with flexion of the middle and distal phalanges of the toes, from paralysis of the interossei.

The *Peroneus longus* (musculo-cutaneous nerve, from external popliteal of sciatic) everts the foot, lowering the inner border, narrowing the foot, and increasing the plantar arch. It also turns the whole foot out on the axis of the leg. It has a very feeble power of extending the ankle. It keeps down the inner part of the foot during extension by the calf muscles, as in walking. In paralysis of this muscle the inner part of the front foot is not supported during extension, and yields to slight force. The foot becomes adducted and rotated, so that the sole is directed inwards, in consequence of the unopposed action of the sural muscles. The inability to press the inner part of the ball of the foot firmly against the ground leads to over-action of the flexors of the great toe. The plantar arch is lessened; there is "flat foot."

Flexors of the Foot.—The *Tibialis anticus* (anterior tibial branch of ext. popliteal nerve) produces simultaneously three movements: it elevates the inner part of the front foot (opposing the peroneus longus); it flexes the ankle-joint, and adducts the foot. The *Extensor longus digitorum* (ant. tibial nerve), besides extending the toes, flexes and abducts the foot. The abduction is in consequence of the outward position of its tendons beneath the annular ligament. These two muscles together produce direct flexion of the foot, or flexion with adduction or abduction, as the force of one or the other preponderates. Paralysis of either weakens flexion, and the corresponding lateral movement is lost, flexion being accompanied by the deviation effected by the muscle that remains. The defect in flexion is greatest in paralysis of the tibialis, and the loss of the instinctive flexion, when the leg is brought forward in walking, causes the foot to catch against the ground. Paralysis of the flexors is followed by secondary contracture of the extensors, and talipes equinus results (Fig. 29), which is the greater the longer the palsy has lasted (Fig. 30). Its occurrence is facilitated, in many cases of palsy, by lessened growth of the bones of the leg, so that the ball of the foot only touches the ground when the foot is extended. There is usually slight

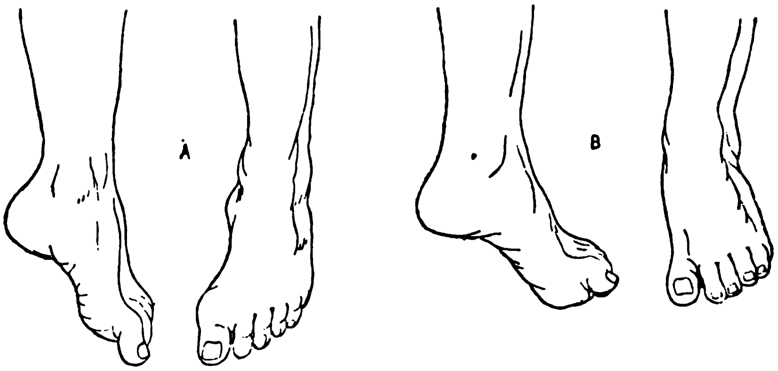

Fig. 29.—Talipes equinus, due to atrophy of the tibialis anticus and secondary contracture of the calf muscles. In A the foot is shown at rest; there is slight equino-varus. In B it is shown during flexion, and the varus is changed to valgus by the action of the peroneus longus. Note the increased extension of the toes in B from the compensatory over-action of the long extensors of the toes. (After Duchenne.)

Fig. 30.

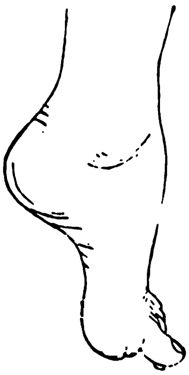

Fig. 31.

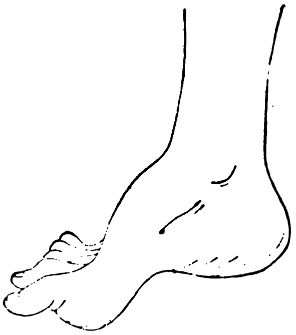

Fig. 30.—Extreme talipes equinus from old-standing palsy of the tibialis [illegible] (infantile paralysis) and extreme contraction of the calf muscles. No [illegible] movement was possible.

Fig. 31.—Paralysis of the interossei and the adductor and short flexor of the [illegible] The first phalanges are over-extended and the second are flexed, [illegible] of the sole is increased. (After Duchenne.)

[illegible] the foot at rest, even when the tibialis is paralysed (see Fig. [illegible] is produced by the sural extensors (p. 42); but in this case the slight valgus at rest is changed to varus on an attempt to flex the ankle (Fig. 29, B).

The *Peroneus brevis* (musculo-cutaneous [peroneal] branch of ext. popliteal nerve) abducts the foot and rotates it, raising the outer edge.

The *Tibialis posticus* (posterior tibial nerve from int. pop.) adducts the foot and curves it, rendering the outer border and instep more convex. Its power of adduction is greater than that of the tibialis anticus, and it does not rotate the foot in the same manner.

These two muscles alone have the power of adducting and abducting without flexing or extending; and in their paralysis these simple movements are lost. If one only is paralysed, a deformity develops corresponding to the action of the other muscle,—talipes valgus in paralysis of the tibialis posticus; t. varus in that of the peroneus brevis.

The *muscles moving the toes* present, in their mode of action, a close correspondence to those of the fingers.

The *Extensor longus digitorum* and the *Extensor longus pollicis* (anterior tibial nerve) extend chiefly the first phalanges, while the *Flexor longus digitorum* and *Flexor brevis* (posterior tibial nerve) flex the last two phalanges. The *Lumbricales* and the *Interossei* (post. tibial nerve by ext. and int. plantar), together with the *Abductor* and *Flexor brevis minimi digiti*, oppose both the other extensor and flexor muscles, flexing the first phalanx and extending the others. This action is of great importance in walking, since they give the last propulsion to the body as the ball of the foot leaves the ground. The *Abductor, Adductor*, and *Flexor brevis pollicis* (plantar nerves from post. tibial) have a similar action on the great toe, but with adduction or abduction respectively. The interossei also produce a lateral movement of the toes, but this action is of little practical importance. In paralysis of the common extensor of the toes, and of the proper extensor of the great toe, the tonic force of the interossei and analogous muscles produces persistent flexion of the first phalanges and extension of the others. If the conditions are reversed, and the latter muscles are paralysed, the first phalanges are over-extended, sometimes even subluxated, and the two other joints are flexed, so that a claw-like form of foot is the result (Figs. 31 and 28). The final propulsion in walking, above described, is much interfered with, and the attempt is painful because the ends of the toes are turned towards the ground.

Other examples of the effects of paralysis of the muscles of the arm and leg are given in the illustrations to the chapter on Infantile Paralysis (Acute Polio-myelitis).

PART II.

DISEASES OF THE NERVES.

GENERAL PATHOLOGY.

Structure.—The individual fibres consist of a central "axis-cylinder," which is the chief functional element, surrounded by the "medullary sheath," or "white substance of Schwann," composed of myelin (M, Fig. 32), a liquid fatty material supported by a fine network of horny substance, "neurokeratin." The myelin is absent in the "non-medullated" fibres of the sympathetic. A delicate membrane surrounds the white substance, the "primitive sheath," or "neurilemma," or "sheath of Schwann" (S). Nuclei (n) lie at intervals within the sheath, between it and the myelin. The white substance is interrupted at regular distances by what are termed "nodes,"—sometimes, from their discoverer, "nodes of Ranvier" (N). The end of each portion, or "internode," is enclosed by the sheath, through which the axis-cylinder passes. Between the incurved extremities of the sheath is a little clear cementing substance, shown by the fine dotting in the figure. There is one nucleus (n) to each internode, about its middle; hence they are sometimes called "internodal nuclei." Around the nucleus is a little protoplasm, and it is probable that a very thin layer of protoplasm everywhere lies between the sheath and the myelin. Each internode may be conceived as a cell, a fat-cell according to Ranvier, consisting of membrane, nucleus, protoplasm, and fatty matter, the cells being arranged end to end, and the axis-cylinder passing through them, as a string passes through a series of tubular beads. The internodes are shorter towards the termination of a nerve. It is important not to confound the nodes with other, imperfect divisions, sometimes termed the "incisions of Schmidt" (IS, Fig. 32, I). These are oblique, incomplete divisions of the white substance. Many exist in each node. They are believed

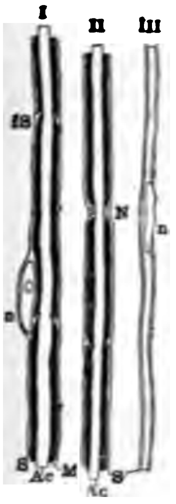

FIG. 32.—Diagram of the structure of nerve-fibres. I and II, medullated, III, non-medullated fibre; S, sheath; M, myelin, or white substance of Schwann; Ac, axis-cylinder; n, nucleus; N, node; IS, incision of Schmidt. (From a preparation of the nerve-fibre of a frog, stained with osmic acid.)

by some histologists to be of artificial origin, but their uniform character seems conclusive evidence that they depend on structural conditions.

The myelin, Ranvier suggests, must protect the axis-cylinder, since its almost liquid consistence will diffuse pressure on the nerve. It may also have an insulating action, which, while not essential for conduction, may render conduction more perfect, and possibly more rapid. The nodal segmentation keeps the almost liquid myelin uniformly distributed along the fibre, and permits nutrient material to reach the axis-cylinder.

The "grey fibres," "non-medullated fibres," consist of an axis-cylinder, sheath, and nuclei (Fig. 32, III), but contain no myelin. They constitute the sympathetic nerves, but some (probably sympathetic fibres) are found in all the spinal nerves. They are absent from the nerves of special sense, except the olfactory, which contains no other fibres.

The nerve-fibres are united into "fasciculi" by delicate nucleated connective tissue, and these fasciculi are similarly connected into larger bundles, while the whole nerve is surrounded by a dense connective-tissue sheath. From this a very delicate "sheath of Henle" extends on to the single nerve-fibres at the final distribution of the nerve. All these tracts of connective tissue are continuous. In them the blood-vessels run and nerve-fibres are distributed. These "nervi nervorum" are derived from the nerve they supply.* Each nerve ("including its sheath") is thus part of the area of distribution of its own fibres.

PHYSIOLOGY OF NERVES.—The first great fact to be kept in view is that the axis-cylinder of each nerve-fibre is the prolonged process of a nerve-cell, which passes gradually into the axis-cylinder, without any abrupt or visible change in structure. This unity explains the secondary degeneration described in the next section; the axis shares the life of the cell, and cannot maintain a separate existence. Further, the fibre shares all modifications in the nutrition of the cell, manifesting it perhaps in a different way, but never preserving a perfectly normal state if the nutrition of the cell is changed.

In the function of nerve-fibres we can see another aspect of the same fact. We speak of cells producing and of fibres conducting what is termed nerve-energy. But fibres also can produce nerve-energy. The stimulation of a fibre is sufficient proof of this. Moreover, if we pass up to the nerve-cell we are met by the necessity of recognising the fact that this function of the fibre is not peculiar to it. The process of the cell must conduct, and the cell must itself conduct. Each motor cell of the cord is part of the path through which the nerve-energy from the brain passes. That which passes through must be conducted, however it may be changed in amount. Even if it is renewed, we cannot so distinguish this function of the cell from that of the fibre as to say that there is not conduction. Nor can we stop here. A fibre can generate nerve-energy, and a cell can conduct: does the whole function of the two differ except in degree? If they are of the same nature, and can perform the same function, we seem unable to avoid the conclusion that the nature of the function must be the same, and that the difference we recognise must be in degree.

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* Horsley, Roy. Med. and Chir. Soc., January 22, 1885.

The great contrast between them is not in function; it is in vitality. Function differs only in degree, just as their structure differs in degree. But the cell possesses an independent vitality, and with this it possesses also that mysterious body—a nucleus. Fibres have nuclei in abundance outside the axis-cylinder; they have no nucleus within their functional element. We seem to have in this feature a vital difference, pregnant alike with meaning and effect.

Further, so far as is yet known, the cells within the central nervous system, from which fibres spring, are those from which they conduct. Hence the direction of conduction is the same as that of degeneration. But this relation does not exist everywhere; it is so in the motor nerves, but the sensory peripheral nerves degenerate downwards and conduct upwards. Here, however, the conditions are manifestly exceptional. Their vitality depends on the cells of ganglia on the posterior roots. These are vital as far as we can see alone; they are not related to function. The function is towards, not from them. The impulses come from the structures in which the nerves end—structures that seem to be developments of the axis-cylinder, far lower in nature than nerve-cells, but possessing the same power of generating an impulse when some form of energy acts on them from without.

These upbearing nerves end peripherally in most cases by dividing into many branches. Thus each fibre is brought into much more extensive relation with the external forces than would otherwise be possible, and may end in an amount of receptive material not incomparable to that of a nerve-cell itself.

The endings of the motor nerves are those to which impulses proceed, and hence have no relation to the considerations just mentioned. These structures in some way transfer the nerve-energy to the muscular protoplasm, at least it disappears in making the muscle contract; and the only fact with which we are immediately concerned is that this must involve continuous molecular contact. In such continuity we probably have the means through which the nutrition of the muscular fibres is influenced by that of their nerves.

LESIONS OF NERVES.—*Secondary Degeneration.*—As we have seen, a nerve-fibre undergoes the structural changes known as "degeneration" whenever it is separated from the cell from which it springs. We have also seen that, as a rule, the degeneration is in the direction of conduction, *i. e.* the cell from which the nerve-fibre conducts is that which governs its nutrition, with the exception of the sensory fibres in the peripheral nerves. The degeneration is commonly termed "secondary" because it is dependent on a "primary" lesion of another kind—as division of the nerve. Degeneration also follows many slighter lesions of a nerve-compression, over-extension, and the like; but it is not certain that this is always the same as that which follows a total lesion. The secondary degeneration is often called "Wallerian," from the name of the pathologist who first studied it. It is of great importance, practical and theoretical. The medullary sheath breaks up into segments, and these into smaller and smaller fragments, and the minute globules and granules are ultimately removed from the nerve-sheath. This is then empty, the axis-cylinder having also perished during the process. The nature of this process of destruction has been studied by Erb and others, but has been chiefly elucidated by the researches of Ranvier, and must be considered in some detail. Ranvier has shown that it is not a mere process of death or decay, but an active process, a destruction of the nerve as such by the protoplasm and nuclei of the internodal cells that constitute it.

The nature of the process has been chiefly studied in animals. The most important facts are illustrated in Fig. 33, in which the examples have been selected from Ranvier's figures and reduced to one tint. In the rabbit the first changes are to be perceived at the end of twenty-four hours. The nuclei are increased in size (A *n*, B *n*); the amount of protoplasm about them is greater than normal, and is granular; there is in places a local increase in the amount of protoplasm within the sheath, compressing the myelin (A *x*, B *x*, C *x*). The nuclei then become detached from the sheath; the protoplasm everywhere increases, and encroaches on the myelin, until here and there it meets across the tube, completely separating the myelin (A, lower part; B, upper part), and with the myelin the axis-cylinder (D). This process then goes on with increased rapidity; the myelin is broken up into smaller and smaller fragments (C, E, F) which are globular in the watery protoplasm, just as oil forms globules in water. The nuclei meanwhile continue to enlarge, and then divide, first the nucleolus and then the whole nucleus (F). The two nuclei may again divide, until (as in G) there are four or more nuclei in each internode, instead of one only as in health. The small globules of fatty myelin seem to become changed in chemical composition, since they are stained less deeply by osmic acid. Ranvier suggests that their fatty matter may undergo a process of saponification. Ultimately they seem to pass through the sheath, are taken up by connective-tissue cells and lymphatic cells in the vicinity (as in J), and are gradually, for the most part, removed. By the time the myelin is in small globules the nuclei cease to multiply. On the removal of the products of degeneration the sheath shrinks, and looks empty in places, but here and there it is enlarged by the nuclei, protoplasm, and a few remaining myelin globules (H). Hence in transverse section many small sheaths are seen with a few of larger size where they have been cut across at these swellings.

In the rabbit the first changes are visible at the end of twenty-four hours; the first complete interruption of the myelin and axis-cylinder occurs about the end of the second day; the process of destruction is

FIG. 33.

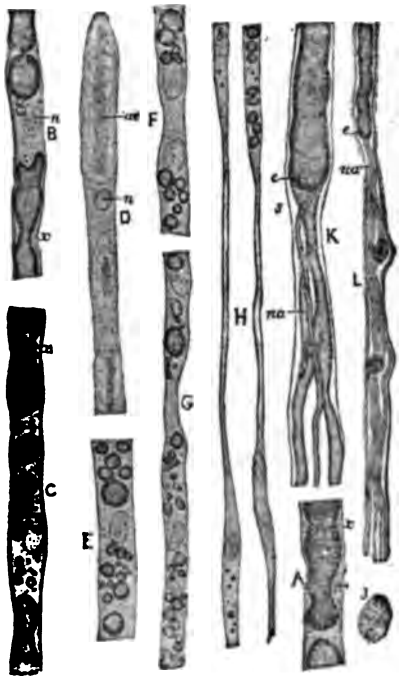

FIG. 34.

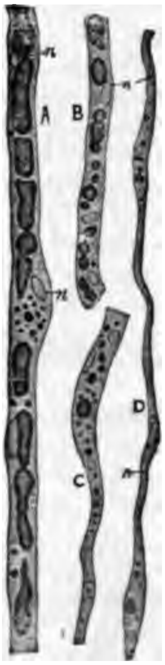

FIG. 33.—A–J, Degeneration of nerve-fibres (osmic acid and carmine staining). A, from sciatic of rabbit four days after section; B, C, same, fifty hours after section; D, a fibre stained with carmine only, to show the axis-cylinder; F, G, fibres from pigeon three days after section; H, two fibres from pneumogastric of rabbit six days after section; J, a lymphatic cell from interfibrillar connective tissue, containing globules of myelin that it has taken up. In all the figures *n*, *n*, nuclei; *c*, *c*, constrictions of the myelin produced by the growth of the protoplasm; *ac*, axis-cylinder.

K, L, Regeneration of nerve-fibres. K, from pneumogastric of rabbit seventy-two days after section; L, from sciatic of rabbit ninety days after section; *e*, rounded end of white substance of central end of nerve; *s*, sheath; *na*, new axis-cylinder. In L are two globules of myelin remaining from the degeneration of the old fibre.

FIG. 34.—Degenerating fibres from cutaneous nerves of man. (After Pitres and Vaillard.) A, from near a bedsore in a case of fracture of the skull; B, C, D, from the fifth nerve in a case of neuralgia and ulceration of the lip; *n*, *n*, nuclei. In A the protoplasm and nuclei are increased, and the myelin is breaking up, the processes having proceeded furthest in the neighbourhood of the nucleus in the middle of the fibre; in B the segmentation has gone on to the formation of globules, which in C are, for the most part, small, and many have been removed, so that the fibre is narrow; while in D all the products of degeneration have been removed from considerable tracts of the sheath.

considerably advanced at the end of the fourth day, and is finished, and the multiplication of the nuclei ceases, towards the end of the second week. In peripheral nerves the changes seem to progress from the lesion to the periphery, but within the central nervous system they are said to begin at the same time in all parts of the fibre. It is uncertain to what extent the difference is real or apparent, being simultaneous in the whole extent of the separated portion of the nerve. They progress more rapidly at its peripheral extremity than elsewhere. At the end of the second day, in the rabbit, stimulation of the nerve by electricity no longer causes muscular contraction. Thus the disappearance of the electrical excitability coincides in time with the first complete segmentation of the myelin and axis-cylinder. This interruption explains what is termed the "loss of excitability." It must be remembered that the loss of continuity is not the only cause of loss of excitability. (See p. 52.) If the ends of a divided nerve are brought quickly into perfect apposition secondary degeneration does not occur in animals, and probably not in man.* Simple division appears to be a less serious injury than the extensive displacement of myelin in a contusion.

Thus the process is the result of an active growth of the nuclei and protoplasm of the nerve, *i. e.* of the cellular elements of which the nerve is composed. Why does this occur? The determining cause is the interruption of the axis-cylinder—its separation from the cell of which it is a process. It follows equally destruction of the nerve-cell. Ranvier points out that the destructive growth of the protoplasm, which follows loss of function in the axis-cylinder, suggests that normally its function restrains the vital energy of the cell-elements. But it is possible that changes in the nutrition of the axis-cylinder precede the segmentation.

A process of destructive cell-activity suggests the idea of inflammation. The degeneration may be regarded as a process of parenchymatous inflammation. Several observers have described other indications of inflammation outside the fibres, especially increase of nuclei, and accumulation of leucocytes in the interstitial connective tissue, and even in the nerve-sheath, and also dilatation of the blood-vessels. Such changes are intense at the primary lesion. Their degree in the nerve below the lesion is very variable, and seems to be proportioned to that in the primary process. This is another mode of stating the important fact that *the irritative character of the secondary process is determined by the irritative nature of the primary disease.*

The process of secondary degeneration occurs more slowly in the rabbit than in a bird, and seems to be still slower in man, in whom it is probable that complete segmentation does not occur until between the fourth and eighth days. It is certain that an identical process occurs in man. Changes in peripheral nerves near bedsores and in the fifth nerve, found by Pitres and Vaillard, are shown in Fig. 34, a comparison of which with Fig. 33 will show the identity of the process. It is highly probable that after complete division of a nerve in man the

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* See Bowlby, 'Injuries and Diseases of Nerves,' p. 32.

changes are the same as in animals. But the most common lesion in man is neuritis, and in this the process of degeneration is probably greatly modified by the severity of the primary lesion; in some cases it occurs rapidly, in others very slowly. In the latter cases, which are chiefly those of focal neuritis, the process cannot be the same as that which follows division of a nerve. The change in irritability, which will be more fully described in the account of the symptoms, is a slow depression, sometimes moderate in degree. The depression may be preceded by an increase in irritability. In such cases there can be no complete segmentation of the nerve-fibres. There must be a gradual alteration in the molecular nutrition of the axis-cylinder, changing its excitability. Even in severe cases there is not usually a sudden loss of irritability; the current necessary for stimulation has to be made stronger, until at last in seven or eight days the strongest endurable current fails to cause muscular contraction. But we cannot infer, from this alone, that there is an actual interruption of the axis-cylinder. A stronger current might still excite the nerve, because, as we shall presently see, when a nerve is being regenerated, an axis-cylinder may conduct, and still not be excitable by currents of ordinary strength.

All severe changes in the nutrition of the fibres involve the intramuscular nerve-endings in the same degree. The evidence of this is that the faradic irritability of the muscles (which depends on the nerves within them) presents changes quite similar to that of the nerve-trunk. But this is not always true in slight changes of nutrition of the nerve. We shall presently see that the slight alterations of irritability in the nerve and muscle do not always correspond. The nerves terminate in structures of special nature, and these may well have some slight degree of nutritional independence.

Regeneration may occur in the nerve after the degeneration is over. It is a slow process, occupying the second, third, and fourth month after division. According to Ranvier, it occurs always by the growth of new axis-cylinders from the central end of the nerve (see Fig. 33, K and L), which ultimately become covered with myelin. One or more new fibres may spring from each central fibre, and these may subdivide. All are enclosed in a sheath which is continuous with that of the central end (Fig. 33, K, *s.*). We must assume that only some of these axis-cylinders persist and achieve functional permanence. Sometimes these fibres twist about, and even turn back and grow upwards, probably in the direction of least resistance. In animals, new fibres may grow through a considerable extent of cicatricial tissue between the divided ends of a nerve, but in man it is doubtful whether regeneration of a divided nerve occurs unless the extremities are brought in contact, or at least close proximity. Some investigators believe that there is a formation of fibres in the peripheral extremity independently of the growth of new fibres from the central end.* In cases of slight injury, regeneration occurs more readily; in these it is probable, as we have just seen, that degeneration has been incomplete. The regenerated nerve-fibres regain some conducting power while they are still much narrower than normal, and before they can be excited by electricity.

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* *E.g.* by Neumann, Mayer, &c. A full abstract of their observations is given

Muscles.—The degeneration of the motor nerves is attended by changes in the nutrition of the muscles. These commence in or after the second week. The muscular fibres become narrower, and may be reduced, ultimately, to one-third of their former width. An increase in width has been said to precede the narrowing for a few days.[*] The transverse striation becomes less distinct, and the striæ seem to be nearer together than in health. The fibres may become cloudy or granular, but do not present actual fatty degeneration except in some very acute cases. If no regeneration of the nerve takes place, the transverse striation gradually disappears, and may be replaced by a longitudinal striation, or the fibres may undergo certain chemical changes, and present a peculiar glassy appearance, which has been called "vitreous degeneration." During the progress of the changes in the fibres, the nuclei of the sarcolemma and of the interstitial tissue are increased in number, and cellular elements, either newly formed, or migrated from the vessels, develop into fibres, so that ultimately the muscular fibres are separated by considerable tracts of connective tissue, and a state of cirrhosis results. If regeneration of the nerve occurs, the muscular changes are arrested, and the normal condition of the fibres is slowly restored. The amount of connective tissue in the muscle is often permanently increased, but the fibres are narrowed out of proportion to the increase in the connective tissue, and this undergoes contraction so that the muscle is for a long time smaller than normal, and its natural bulk may never be regained. If no regeneration of the nerve occurs, the muscular fibres gradually disappear; fibrous tissue takes their place, and, slowly contracting, permanent shortening may result. Similar shortening sometimes occurs when there is partial recovery of the nerve and muscle. In most lesions of nerves, other than actual division, some fibres recover, even though others are permanently

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by Bowlby (loc. cit.), and also by Allen Starr in the Middleton Goldsmith Lectures for 1887 ('New York Med. Record,' February, 1887). By some the new axis-cylinders are said to be formed by elongation of the fragments of the old axis; by others from the nuclei of the sheath of Schwann, and appearances have been seen by Bowlby (loc. cit.) in man which seemed to him to bear this interpretation. But Ranvier's careful investigations have been fully confirmed by the minute researches of Vanlair ('Arch. de Biologie,' 1885), and it is difficult to understand that perfect axis-cylinders should be formed and remain unexcitable, as the peripheral segment certainly does. Structures may be formed resembling axis-cylinders that are not really capable of the proper function. The weight of Ranvier's undisproved and confirmed observations is very great. It is possible that the process, in the peripheral part of a nerve, is influenced by the connection with other nerves and the recurrent influence of anastomosing fibres.

[*] Steinert, 'Verh. Phys. Ges. Wurzburg,' 1858, No. 10.

destroyed. The muscular degeneration is the result of that of the motor nerves, as described at p. 22.

Symptoms of Nerve-injury and Degeneration.—The symptoms that attend the lesions of motor nerves and the consequent degeneration are of great importance. The lesion of the nerve causes paralysis of the muscles supplied by it, due to, and in proportion to, the interference with the conducting power of the nerve. The muscles at once become flabby from loss of tone, and to this atony actual wasting is added in the course of a few weeks. The wasting is due to the reduction in size of the muscular fibres. If the sensory nerve-fibres are not interrupted, the muscles become tender to the touch, and pain is caused by their strong contraction, due probably to the effect of interstitial inflammation on the sensory nerves, which end in the connective tissue.

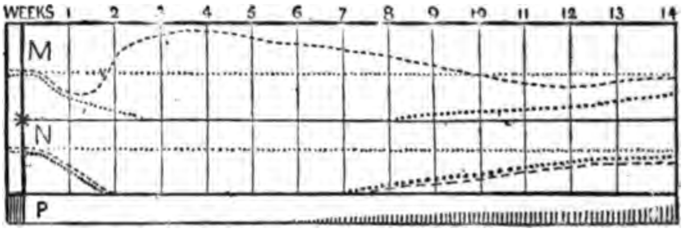

Fig. 35.—Type of degenerative reaction in a case of nerve-injury of moderate severity. (After Erb.) Muscle irritability lowered from middle of first week; faradic, extinct in middle of third, reappearing in ninth; voltaic, increased from middle of second week until tenth, then depressed until fourteenth. Nerve irritability (changed alike to both currents) is lowered from middle of first week, lost at end of second, reappearing at seventh. Power lost until end of fifth week.

In this and the following figures the normal degree of irritability (ascertained from the healthy side) is represented by the finely dotted horizontal line (*n.l.*); faradic irritability, F, by a line of larger dots; voltaic irritability, V, by a broken line; M, muscle; N, nerve; P, power of voluntary contraction, its degree shown by the vertical lines; the asterisk marks the occurrence of the lesion. The vertical divisions represent time intervals. (Many of these indicating letters are given only in subsequent figures.)

The most important symptoms are those that are afforded by electrical examination of the muscles and nerves, since they enable the degenerative changes to be ascertained and followed during life. The alterations in the electrical reaction, consequent on this degeneration, have been already briefly mentioned (p. 24), but must now be described in greater detail. The rapid degeneration of a nerve, which follows a severe lesion, is attended by a loss of irritability on electrical stimulation, the loss being the same to faradism and voltaism. After such lesions as are common in man, neuritis for instance, there is no sudden loss, such as occurs after injury of a nerve in an animal, when the nerve becomes segmented, but there is a more or less rapid diminution of excitability, and this goes on until no stimulation can be produced, even by a strong current. The progressive changes in irritability may be conveniently represented on a chart. Fig. 35 shows the typical course of the changes of irritability in a case of moderate severity. In the muscle (M) a fall of irritability (due to the degeneration in the nerve-endings) occurs simultaneously with that in the nerve-trunk (N), and the faradic excitability becomes extinct at the same time in both nerve and muscle. The fall in voltaic irritability is quickly arrested by the change in the muscular fibres, through which they soon become more excitable than normal to the voltaic current. This change usually occurs during the second week, and the irritability continues to increase during the third and fourth weeks. At its maximum it may amount to three, four, or five cells of the battery *i e.* contraction can be obtained in the paralysed muscles with a current weaker, by so many cells, than is necessary to cause contraction in a corresponding unaffected part. The further course of the changes in irritability depends on the severity of the lesion and the intensity of the degeneration. In a case of moderate degree, such as is shown on the chart, nerve irritability reappears about the end of the second month, usually after some voluntary power is regained. It is at first low, so that a strong current is required. It gradually increases, but for a long time continues a little below the normal degree. This return of nerve irritability is accompanied by a corresponding return of faradic irritability in the muscles (*i. e.* in the intra-muscular nerves). The increase of voltaic irritability often persists long after recovery of power, but it lessens as faradic irritability returns, and, as shown in the chart, it may fall below the normal before it ultimately regains its original degree.

Slight changes in irritability can be ascertained only by a comparison with the corresponding part on the other side in the same individual. Moreover, when we speak of excitability being "lost," we mean that we can obtain no stimulation by any strength of current that can be borne. This strength varies in different persons and in different parts. The more sensitive the part, the earlier irritability seems to be lost, and the later it seems to return; and the greater the resistance of the skin, the stronger must be the strength of current.* In many cases, if we could use very strong currents, the irritability would be found to be merely much lowered, although it seems to be extinct when we can only test it with currents of moderate strength.

If the lesion is very severe, so that there is no recovery, and no regeneration of the nerve, the loss of nerve irritability, and of the faradic muscular irritability, is permanent. The increase in voltaic irritability persists for months, and then gradually falls as the muscular fibres waste, and becomes lower and lower (see Fig. 36)

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* Hence the importance of ascertaining what current is actually passing by the use of a galvanometer, which may be thus used when the comparison with the other side is made by a number of cells, to spare the patient the longer and more painful process which the more exact comparison by absolute current strength involves.

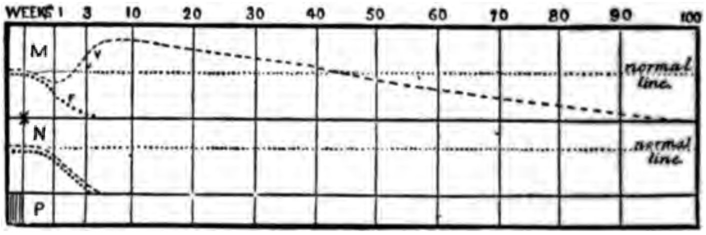

FIG. 36.—Type of reaction in a case of complete and permanent damage to a nerve. (After Erb.) Early course as in the last diagram; no return of power, nerve irritability, or faradic irritability in the muscle. The early increase in voltaic irritability gradually lessens, and at the end of ten months falls below the normal, but is not extinct until nearly two years.

until ultimately no reaction can be obtained, the fibres having perished. It does not become extinct until at least a year has elapsed, and sometimes (as in the chart) only towards the end of the second year. Often, when no contraction can be produced on the first attempt, after two or three applications, distinct contractions are obtained.

The changes we have hitherto considered are in the *degree* of irritability, "quantitative" changes. But the quantitative increase in the muscular irritability is often accompanied by a change in the order of response, according to the pole that is applied and the strength of current—a "qualitative" or "polar" change. We have already considered its general characters (p. 24), and have seen that it consists in an undue readiness of response at the positive pole (Anode) compared with that at the negative pole (Kathode) (Fig. 37, B and C), the muscle being normally the more sensitive to the latter (Fig. 37, A). Writing Cl. for the closure of the circuit, O. for its opening, and C. for contraction, the normal reaction is—

$$1.\ \text{K.Cl.C.};\ 2.\ \left\{\begin{array}{l}\text{An.Cl.C.};\\ \text{An.O.C.};\end{array}\right.\ 3.\ \text{K.O.C.}$$

In disease—

$$1.\ \left\{\begin{array}{l}\text{K.Cl.C.};\\ \text{An.Cl.C.};\end{array}\right.\ 2.\ \text{An.O.C.};\ 3.\ \text{K.O C.: or even}$$

1. An.Cl.C.; 2. K.Cl.C.; 3. K.O C.; 4. An.O C.

This qualitative change is not always present, at any rate in recognisable degree. Even when there is a marked quantitative change, the kathodal closure contraction may still occur first. When the change is present, it is only in the muscles, and it must depend on the muscular fibres themselves. In the motor nerve the kathodal response is always the first, although a qualitative change has been detected in degenerated sensory nerves.

The muscular contractions which occur thus with undue readiness differ from normal contractions, excited through the nerves, in their distinctly deliberate character. Instead of the quick, lightning-like contraction, the movement is distinctly longer in reaching its maximum and longer in its duration. This difference is very well shown in the accompanying tracings of contraction (Fig. 37). Sometimes a slight

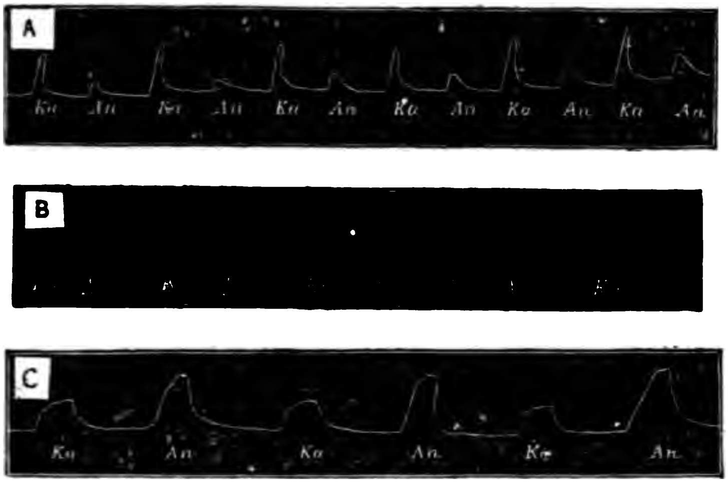

Fig. 37.—Tracings of the muscular contractions in nerve-degeneration. (After Erb.) *Ka*, Kathodal closure contraction; *An*, Anodal closure contraction. A, tracing in health; *Ka* much stronger than *An*; contractions sudden. B, tracing in nerve-degeneration with moderate current. *An* much greater than *Ka*, the latter scarcely visible. Contraction slower, shown by the more sloping upstroke. C, the same, with a stronger current. *Ka* greater, but still less than *An*; the slow character of the contraction and its long duration well marked.

abnormal tetanic contraction during the passage of the current also occurs. During the period of increased voltaic irritability the mechanical excitability of the fibres is often increased. If they are directly percussed, they respond with a distinct slow contraction (Erb).

Such are the changes in irritability which occur in cases of nerve-lesion and degeneration of moderate and considerable degrees of severity. Certain variations are occasionally met with, and these occur especially in cases of neuritis of slight degree. In severe cases the fall in nerve irritability, which usually commences in the middle of the first week, may not take place until the end of this week, although it may then progress so rapidly that no stimulation can be obtained at the end of the second week. An example of this is shown in Fig. 38.[*] The same chart, and some of those that follow it, illustrate another very common variation from the type above described. There may be no fall in the voltaic excitability of the muscles before the commencement of the degenerative increase. Indeed, this initial fall is as often absent as present.[†] The change in the nutrition of

[*] This and the following charts are from cases of neuritis of the facial nerve.

[†] The fall *seems* to suggest that the voltaic irritability of the muscular fibres is normally below that of the nerve-endings, and this is intrinsically probable. The presence or absence of the fall may depend on the rapidity with which the nerve-endings degenerate. Some facts, moreover, that have come under my notice, suggest that the motor nerve-endings possess some nutritional difference from the fibres, in consequence of which the relative condition of the two, especially as regards excitability, varies in different cases, especially in the early stage.

the muscle may then coincide with, instead of succeeding, the degeneration of the nerve-endings (Figs. 38, 40). Sometimes, if the degeneration is rapid, there is an interval of a few days before the secondary changes in the muscle have reached the degree necessary to produce increased irritability. In a slight degree of degeneration, the increase in the voltaic irritability of the muscles may be postponed for a week or more after the nerve irritability begins to fall, and until the rise occurs the voltaic irritability of the muscles may remain normal, even when their faradic irritability falls with that of the nerve-trunk. An instance of this is shown in Fig. 39.

Fig. 38.

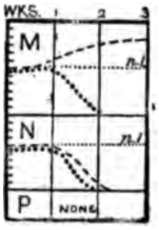

Fig. 39.

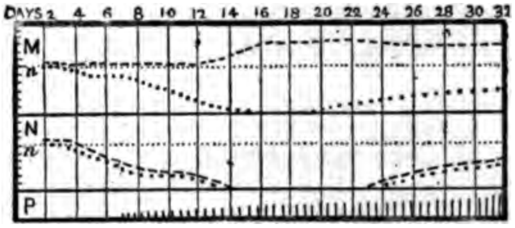

Fig. 38.—Severe neuritis, paralysis complete for months; recovery imperfect. Irritability of nerve normal during first week; fell rapidly during the second; lost at its close. Simultaneous increase in voltaic irritability. The divisions on the left side represent cells of the voltaic battery, and half-centimetres of the secondary coil of Stöhrer's induction apparatus, in this and the following charts.

Fig. 39.—Slight neuritis; slight return of power on seventh day, slowly increasing; normal in fifth week. Muscle: faradic irritability lessening from fourth day, lost on fifteenth, reappearing on twentieth. Voltaic irritability normal till twelfth day, then augmented. Nerve-trunk: lowered from fourth day without initial increase, reappearing on twenty-third day; changed alike to faradism and voltaism.

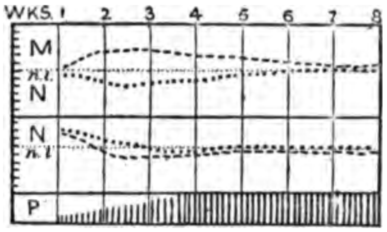

Fig. 40.—Slight neuritis; power not entirely lost, and becoming normal during the third week. Slight deg. react. in muscle developed during second week, and continued till seventh. Nerve irritability increased during second week, passing during the third into transient depression, the voltaic irritability falling more than the faradic.

When the nerve-lesion is very slight in degree, the fall in nerve irritability may be preceded by a rise, which may be far greater in degree than the subsequent fall. The fall may indeed be not only slight, it may be altogether absent, so that the rise constitutes the only symptom. This initial increase in irritability is of considerable interest as the manifestation of the slightest degree of alteration in the nutrition of nerve-fibres.* It may last for a few days or for two weeks (Fig. 40), and I have once known it to continue for five weeks (Fig. 41). Although the

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* A similar change is met with in some central diseases, as chorea and paralysis agitans.

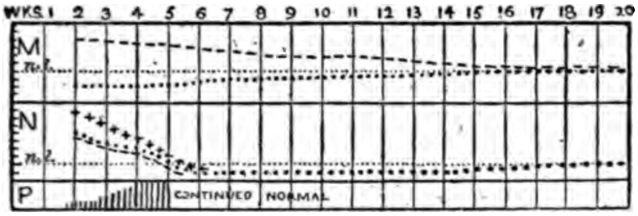

Fig. 41.—Slight neuritis; quick recovery of power during third and fourth weeks. Degen. react. in muscles present at the end of the second week, slowly lessening, but present in slight degree until the fourth month. Considerable increase in irritability in the nerve-trunk at the end of the second week, greatest to the faradic shock (crosses) and lasting until the fifth week.

change in irritability of the nerve is usually the same to both currents, a partial exception to this rule is presented by the increase in irritability we are now considering The change is not always quite equal to faradism and to voltaism, and it is frequently much more marked to the isolated faradic shocks than it is to either the voltaic current or the rapid succession of shocks that constitutes the faradic current. This is shown very strikingly in Fig. 41, and in less degree in Fig. 43. In the former the irritability to the two currents was the same; in the latter that to voltaism was distinctly the greater. I have once met with a slight but distinct and prolonged diminution in faradic irritability when no change could be found to voltaism (see Fig. 42). Bernhardt has observed lessened irritability to faradism

Fig. 42.

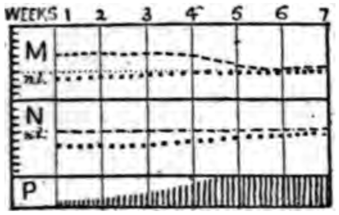

Fig. 43.

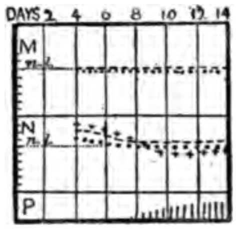

Fig. 42.—Slight neuritis; power regained during the second week. Deg. react. in muscle distinct on eighth day, lessening during second and third weeks. Nerve: lowered faradic, normal voltaic irritability.

Fig. 43.—Very slight neuritis. No change in muscular irritability; irritability of nerve-trunk increased from fourth to eighth day, most to faradic shock (crosses), least to faradic current.

with distinct increase to voltaism in an ulnar nerve the seat of traumatic paralysis. In one singular case of double facial palsy, with weakness of the limbs, and changes in their muscular irritability, the voltaic excitability of the facial nerves was not lessened, although faradic irritability was lost to such a current as could be borne. The case was probably one of non-alcoholic peripheral neuritis.*

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* The observations were made with great care and with full knowledge of a

Moreover, the law that the faradic irritability is changed in the same degree in the muscles (nerve-endings), and in the nerve-trunk, is also not always true of slight cases. The muscle-nerves may present no change when the excitability of the nerve-trunk is considerably increased (Fig. 43). The probable significance of this difference has been already mentioned (pp. 52 and 57, *note*).

The electrical excitability of a nerve and its power of conduction are usually lost together. But when a nerve is recovering from a local lesion, and regeneration is in progress below the lesion, some power of conducting the voluntary stimulus may return in the peripheral portion before it becomes excitable by any form of electricity. This seems to show that the nerve-fibres can conduct, that is, pass on the nerve-force they receive as such, before they can produce nerve-energy in response to the influence of a "stimulating" force acting on them. Another rare anomalous condition has been noted by Bernhardt and Grünberg, in recent cases of nerve-injury. When the lesion arrests all conduction of the voluntary stimulus a strong faradic stimulation of the nerve above the lesion may still cause slight contraction in the paralysed muscles. It is probable that this difference is simply one of strength of stimulus, and that the nerve impulse generated by the electricity is stronger than that descending from the brain.

Recovery of the nerve is attended by gradual restoration of power over the muscles, the nutrition of which slowly improves; but if the wasting has been considerable, a long time elapses before they regain their normal size. Indeed, as already stated, they may be always somewhat smaller than the corresponding muscles on the unaffected side. Curious secondary spasmodic symptoms are common after palsy of the facial nerve (*q. v.*)—spontaneous muscular contractions, and associated over-action of the affected muscles. This associated over-action is very rarely met with after palsy of the nerves of the limbs, but coarse, fibrillary, muscular contractions, and a tendency to cramp, are not uncommon, and may persist for years. (See also Neuritis.)

Sensory symptoms also result from lesions of the nerves that contain sensory fibres. The interruption of the fibres arrests conduction, and causes loss of sensation in the part supplied by the nerve, just as it causes motor palsy. But a slight lesion of a mixed nerve may cause persistent muscular paralysis, and sensation be unaffected, or impaired only in slight degree, and for a short time. This result is so frequent that we cannot ascribe it to a difference in the relative damage to the motor and sensory fibres. It must be due either to the fact that the sensory fibres recover more readily, or that a slight degree of conduction may suffice for the stimulation of the sensory centres in the brain and not for that of the muscular fibres. There is also a third hypothesis, and that is a difference in the sensory and motor

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frequent source of fallacy—the diffusion of the voltaic current to the muscles themselves. Erb has observed a similar change after injury to a nerve in an animal.

impulses in physiological character.* In another class of cases, however, we must seek a different explanation of the persistence of sensibility in the area supplied by the nerve. There may be no loss even when a nerve is completely divided. This is observed chiefly near the extremity of a limb. It can only be explained by what is termed "recurrent sensibility." Anastomoses exist between the terminal fibres of different nerve-trunks, and it would seem that, in some individuals, the connection of sensory fibres suffices for conduction. Thus, of two persons in whom the ulnar nerve at the wrist has been divided, one may have total anæsthesia in the fingers supplied by tho nerve, and in the other there may be little or no loss.

The peripheral segments of the sensory fibres degenerate, like the motor nerves. We cannot test them, and ascertain the degeneration in the same way, in severe lesions, because their stimulation cannot be perceived. Nevertheless an altered polar reaction has been observed in slight cases, analogous to that which occurs in the muscles. A sensation is produced with the positive pole by a weaker current than is required with the negative pole. In some cases of nerve-injury and neuritis a delay in conduction of pain may be observed.

Increased sensitiveness in the area of distribution of the nerve, with or without spontaneous pain, is very common in cases of partial lesions of nerves. It must be referred to the morbid changes in their fibres, alterations in the nutrition of the axis-cylinder or nerve-endings, probably analogous to the slight changes in the motor nerves which increase their excitability. Pain and tenderness of the nerve-trunk are also frequent in the same cases, due no doubt to the increased sensitiveness of the "nervi nervorum" distributed in the sheath. The sheath of each nerve is part of the distribution of its own fibres, and those that end in the sheath seem to be very readily deranged. We shall see the importance of this consideration in relation to neuritis and neuralgia. The pains, both local and distant, are sometimes very severe and persistent.

Reflex Action.—All lesions of nerves abolish reflex action in the area of the distribution of the nerve affected. Myotatic irritability is also lost, from damage to either the motor or sensory fibres concerned, and this loss may persist long after the other symptoms.

Vaso-motor and trophic disturbance occurs frequently in consequence of lesions of nerves. The vaso-motor nerves run in the mixed nerve-trunks, and suffer with the other fibres. The general character of the disturbance has already been described (p. 22). When the lesion is acute and irritative, vascular dilatation and an increase of temperature occur at first, and are followed by passive hyperæmia and lowered temperature. There may be œdema, and an increased secretion of sweat. The changes in the skin, acute and chronic, are those that have been already described. "Glossy skin," red, smooth and thin, is especially common, and is frequently accompanied by atrophy of the subcutaneous tissue, and perhaps of the bone, so that the finger-tips become narrow, and present an aspect that is very characteristic. Burning pain, with tenderness, often accompanies the change, and may continue for long. Occasionally, instead of becoming thin, the epidermis increases in thickness, the nails often become thick and rough, and present transverse or longitudinal furrows.* In parts where there is much subcutaneous tissue this may also become thickened and have a peculiar doughy feel, resembling that met with in myxœdema. The growth of the hair may be lessened or increased. Sloughing of the skin is far less common than in central diseases, but vesication occurs with extreme readiness, and sometimes vesicles or bullæ appear to form spontaneously. Ulcers result which erode and destroy the tissues, especially of the finger-ends. Mustard plasters also blister the skin more readily than in health. It is important to remember these facts, because the affection is one in which hot applications are often recommended. A gentleman dislocated his shoulder, and either by the displaced bone, or in the reduction, the brachial plexus was seriously injured. The muscles of the hand wasted, and the skin became glossy. He was advised by a surgeon to bathe the hand daily in hot water. One day his wife bathed it in water which to her was pleasantly warm, and which caused him no discomfort, but the result was that the hand was covered with blisters, and ulcers were left which did not heal for months.

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* The experiments of Leuderitz ('Zeitschr. f. klin. Med.,' Bd. iii) merely confirm the fact, and do not really explain it, as he and others have assumed.

The joints often suffer in their nutrition. Acute inflammation, such as occurs sometimes in spinal affections, is rare, but chronic changes in the joints are frequent, with much tenderness; alterations in the articular surfaces, and fibrous adhesions result, which limit movement. When the muscular wasting is slight, the condition may resemble a primary joint affection, and this mistake in diagnosis is sometimes actually made.

INJURIES OF NERVES: COMPRESSION.

Traumatic lesions of nerves are in the province of surgery. They belong to the physician only in so far as their effects are produced by the agency of a neuritis, the mechanical origin of which may escape notice. Neuritis is described in the next section. A word is needed, however, on the subject of simple, slow compression of nerves, not because the symptoms are grave, but because they often give rise to very grave mental concern on the part of the patient, and to occasional doubts in the mind of his medical attendant. Sudden compression bruises the nerve-fibres, and only concerns us as a cause of neuritis. Slow pressure on any nerve, however slight, if maintained for a time, causes a familiar tingling in the region of distribution of its fibres, often with a sense of warmth, with disordered sensations and even a loss of sensibility in the skin. Often this is accompanied by an inability properly to put in action the muscles that are supplied by the nerve. All the symptoms pass away in a few minutes, a sense of cold and an inability to direct movements being often the last effects. If they do not pass away, the persistent effects are ascribed to a neuritis such as is produced by any other cause.

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* Several excellent illustrations of these changes are given by Bowlby, loc. cit., where also a full account of traumatic lesions is given.

But neuritis is not the primary lesion, and the initial symptoms are not produced by the agency of inflammation. Hence, when they persist, it is doubtful whether we should ascribe them to the inflammation unless they occur after an interval. The mechanism by which the tingling and early anæsthesia are produced, probably causes also the lasting symptoms. The compression no doubt causes anæmia, but the known sensitiveness of nerves to mechanical stimulation makes it probable that the effects are chiefly produced by the mechanical influence of the pressure on the elements of the nerve-fibres, and that a compression for a few hours has such an effect in separating the molecular elements of the white substance, as to set up a secondary degeneration of the same character as that which results from division of a nerve. The neuritis is thus no more the cause of symptoms in compression than in division. In each there is a displacement of molecules interrupting conduction. This is also the conclusion of Weir Mitchell, who found that a pressure of eighteen to twenty inches of mercury for twenty seconds abolished the power of conduction, though only for a few minutes, in spite of conspicuous change in the white substance. The latter is still greater, and the effects more enduring, in a contusion.

The cause of the tingling in the region of distribution of the sciatic nerve is recognised when it is due to pressure by sitting. But it is otherwise when the pressure is on the nerves of the arm and is produced during sleep. The tingling may be felt in the region of the ulnar nerve, in consequence of flexion of the elbow, or in the whole hand from the pressure on the nerves of the brachial plexus on the side on which the patient is lying. Waking up with the tingling, and sometimes with cramp or powerlessness in the hand, he is naturally alarmed. The diagnosis, however, is rendered easy by the unilateral character of the symptoms, and by the fact that they are always felt on the side that is bearing the weight of the body. The symptoms are produced much more readily when the general health is bad; they last longer and more often end in neuritis. The pressure of crutches on the brachial plexus may have the same effects.

INFLAMMATION OF NERVES: NEURITIS.

Neuritis, or inflammation of nerves, presents various characters, which have led to the distinction of numerous forms. The inflammation of single nerves usually begins in the outer sheath of the nerve, and this has been distinguished as "perineuritis." It may involve also or chiefly the connective tissue between the bundles of nerve-fibres ("interstitial neuritis"), or the nerve-fibres themselves ("parenchymatous or degenerative neuritis"). Some forms of neuritis have a tendency to ascend the nerve, and this variety has been termed "ascending neuritis" or "neuritis migrans." One nerve-trunk may be primarily affected, or many nerves may suffer at the same time. The latter condition, "primary multiple neuritis," is of great importance, and will be described in a separate section.

Most forms of neuritis may be either acute or chronic. Acute neuritis usually subsides into a chronic stage, and its symptoms may thus persist for a long time. Other varieties that have been distinguished depend upon the cause to which the inflammation is secondary. Thus we have cancerous, syphilitic, gouty, diabetic, and other forms.

Causes.—Injury causes more or less inflammation which occurs readily, and follows very slight damage. Neuritis may thus be set up by all sorts of wounds, by contusions, by compression from without or by adjacent stuctures, and by over-extension of the nerves. Those nerves that pass by joints are liable to injury from dislocations, either by the displacement of the bones, or during reduction. In fractures the nerves may be injured directly, or may be compressed by the callus that is formed. Nerves are sometimes damaged by a violent contraction of the muscles through which they pass. It is probable also that muscular effort excites neuritis in other situations, especially in persons who are predisposed. Moreover, a strain on the fibrous tissues of joints, fasciæ, &c., may set up inflammation in them, which may spread to the nerves.

Neuritis may arise by extension from adjacent inflammation. The nerves near suppurating joints may be involved, and even, it is said, those that pass by a joint the seat of simple inflammation. The latter is certainly rare. The intercostal nerves have been affected by extension from an inflamed pleura. The cranial nerves and the spinal nerve-roots are involved by extension from the membranes.

Exposure to cold is another cause of neuritis. It is then often called "rheumatic." The nerve, or rather its sheath, may be primarily affected, or the first effect may be inflammation of the fasciæ, from which the inflammation spreads to the nerve-sheath. Persons who are liable to muscular rheumatism, and those who are gouty, suffer thus with especial frequency. But exposure to cold also acts in another way, by causing a blood-state which acts on many nerves at the same time. Many general diseases have a similar power of causing either an isolated or a multiple neuritis. It is through this agency that diphtheritic paralysis is produced, and the same effect may be due to the presence of other toxic material in the blood, metallic poisons, alcohol, &c., an account of which will be found in the section on "multiple neuritis." When an isolated neuritis occurs during an acute disease it is probably produced through the agency of compression, unnoticed during the prostration. Neuritis has also been found in the vicinity of bedsores (Pitres), and though probably due to pressure may be part of the mechanism by which they are caused. Some chronic general diseases cause only isolated neuritis,—either simple, as that which is due to gout (when the general disease must be regarded chiefly as a predisponent); or special, as syphilitic, cancerous, or leucocythæmic neuritis, in which there is an infiltration of the nerve with the special tissue-element characteristic of the general disease.

The characters of the simple isolated form constitute the chief subject of this section.

Pathological Anatomy.—The changes differ according as the inflammation affects primarily the adventitial connective tissue, "adventitial neuritis," or the fibres themselves in the "parenchymatous" form. Isolated neuritis is generally adventitial, and will be first described. In acute inflammation the affected part of the nerve is red and swollen. The redness depends on distended vessels, which may be visible on the surface. The swelling is due to œdema, or to a sero-fibrinous exudation, sometimes jelly-like in aspect. The microscope shows leucocyte-like corpuscles surrounding the vessels, infiltrating the sheath (Fig. 44) and accumulating between it and the nerve. There may be even small extravasations of blood. These changes may be limited to the sheath in what is called "perineuritis," or may extend into the substance of the nerve in "interstitial neuritis." In the latter case the lymphoid corpuscles infiltrate the septa, and may even be seen in the substance of the fasciculi, between the nerve-fibres. These changes may be continuous along a considerable tract of the nerve (diffuse neuritis), but more frequently they are chiefly marked at certain places, which are separated by por-

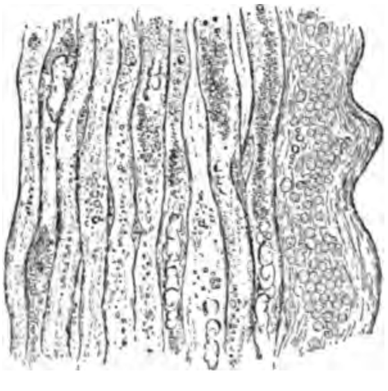

Fig. 44.—Neuritis: degeneration of nerve-fibres, the myelin broken up into masses, globules, and granules. Accumulation of leucocytes in nerve-sheath. From a case of multiple neuritis. (After Leyden.)

tions of the nerve which are so little affected as to appear to the naked eye to be normal ("focal" or "disseminated neuritis"). The foci of inflammation are chiefly situated at places where the nerve turns round a bone, or emerges from canals or fasciæ, or divides.

The extent to which the nerve-fibres suffer varies much. They usually present little change when the inflammation is limited to the sheath, unless the nerve lies in a bony canal, or in rigid fibrous tissue, within which the sheath cannot expand; its swelling then exerts pressure on the fibres. When the inflammation is interstitial the fibres suffer more readily, although not invariably. On the other hand, they are sometimes found much altered, when the connective-tissue elements are but little affected. In this case the inflammation is "parenchymatous" and begins in the nerve-fibres, the interstitial tissue being secondarily involved or unaffected. The changes in the fibres are almost the same as in degeneration (Figs. 33 and 34). It was pointed out how closely the two processes of inflammation and degeneration are connected, and how difficult it is to separate, or even to distinguish them, in many cases. The myelin of the white substance first breaks up into segments, more or less elongated, often with smaller globules of myelin between or beside them. The masses are cloudy or finely granular in aspect. The axis-cylinders are interrupted where the myelin is divided. The nuclei of the sheath are increased in number, and the protoplasm around them is increased in quantity. Next the myelin is divided into smaller globules and granules, and the axis-cylinder is no longer recognisable. The myelin then disappears in parts of the tubes, while it remains at other parts, but is still more finely divided. Lastly, the sheaths become empty and very narrow, containing only nuclei at intervals, with here and there a little finely granular material, or sometimes some brownish pigment granules. The process may thus go on to complete destruction of the fibres. These commonly suffer unequally; fibres that have a normal appearance are scattered among those that are much altered.

In chronic interstitial neuritis the axis-cylinders often suffer less than the white substance. The latter becomes atrophied, so that the fibres are smaller than normal. If the interstitial inflammation is very intense the fibres may break up as above described. As the inflammation subsides, the new cellular elements that have made their appearance assume the aspect of fusiform cells, and fibrous tissue is developed, either from these cells, which become less numerous, or from the intercellular exudation. This fibrous tissue surrounds and encloses the fasciculi, a condition that has been termed "sclerosis of the nerve." A firm fusiform swelling may remain at the affected spot, and this may be adherent to adjacent structures. Sometimes fat is ultimately formed in the new connective tissue, a condition that has been rather unnecessarily termed "lipomatous neuritis" (Leyden); the fatty deposit does not seem to be related to any special form of neuritis.

In syphilitic neuritis, which affects chiefly the cranial nerves, there is a cellular growth in the sheath and interstitial tissue similar to that which constitutes other syphilitic new formations, with a variable amount of alteration of simpler inflammatory character. The growth in the sheath may amount to a distinct syphilitic tumour, with or without interstitial changes. In cancer, nerves adjacent to the new growth may present simple interstitial neuritis, or may be infiltrated by a growth of cancer-elements spreading to the nerve by direct extension. In leucocythæmia there is a dense infiltration of the nerve with leucocytes.

The destructive changes in the nerve-fibres extend down the nerve to the periphery in a process of secondary degeneration, more or less inflammatory in nature, as already described in secondary degeneration. Usually the central portion of the nerve remains free, the alterations ceasing a short distance above the seat of the primary inflammation. In rare cases an ascending neuritis (n. migrans) passes up the nerve, and may spread, at a plexus, to other nerve-bundles and other nerve-trunks. The formation of fibrous tissue in the inflamed part, and the cicatricial contraction of this tissue, often prolong the irritation of the nerve-fibres, and the symptoms due to this and a state of "chronic neuritis" is said to be left.

SYMPTOMS.—The symptoms of neuritis vary extremely according to its intensity, its extent, and the nerve that is affected. In two places, the brachial plexus and the sciatic nerve, the symptoms are sufficiently special to make their separate description desirable, although the general account applies to them also.

The onset of the acute form is sometimes attended by some constitutional disturbance, especially when many nerves are affected. The chief symptoms are local. The most prominent is pain, felt in the inflamed part of the nerve, and also often in the part to which it is distributed (see p. 61). Sometimes the pain involves the whole limb, and in severe cases it may be most intense, burning, boring, rarely darting, in character; usually worse at night; increased by movement, by postures that involve tension or pressure on the nerve, and by whatever causes passive congestion of the limb, such as the act of coughing. Sometimes it seems to radiate into distant parts, and not rarely pain is also felt in the corresponding region of the opposite limb. The sensitiveness of the whole of the affected region is increased, and even the bone may be tender, so that at first attention may not be directed to the nerve, but when this is pressed great pain is always produced. In slighter cases the pain is usually limited to the nerve and its distribution. It may, indeed, be absent when the nerve or branch is chiefly motor, a fact difficult to explain unless the sheaths of such nerves contain fewer sensory fibres than usual. If the nerve is accessible to direct examination it may be felt to be distinctly swollen at the affected part. Occasionally the skin over it has been observed to be red, and rarely there has been slight œdema.

Spontaneous sensations may be felt in the region supplied by the nerve, tingling, &c., and the skin may be hyperæsthetic. After a time, as the nerve-fibres suffer, sensation may be perverted, or even lessened; complete anæsthesia is usually confined to small areas. The muscles supplied by the nerve become weak in various degree, tender, and present fibrillar twitchings; they are seldom powerless, but pain may prevent their contraction. They present the altered electrical reactions characteristic of nerve-lesions. Increased perspiration has been observed in the part of the skin supplied by the inflamed nerve, very rarely it is arrested, and sometimes, although rarely, eruptions occur. Herpes is not common from ordinary acute neuritis. The epidermis may become thickened into a state like ichthyosis, or atrophy as "glossy skin." Effusion into joints has been observed in very rare cases. The constitutional disturbance which may attend the onset subsides in the course of a few days, but the pain and other symptoms usually persist in undiminished severity for several weeks. They ultimately slowly subside into a chronic stage, and may continue as a secondary neuralgia due to a habit of over-action set up in the centre.

In the chronic form constitutional disturbance is absent, and pain is the prominent symptom from the first. The affection of motion, sensibility, and the trophic changes are similar to those met with in the acute form. In both forms the muscles may waste, and present the reaction of degeneration when they are tested with electricity (p. 45). In slight cases the initial increase of excitability in the nerve is often well marked. Trophic changes in the skin are very common; so is alteration in the nutrition of the joints, and adhesions form, limiting the movement, and fixing the parts in the position corresponding to the muscular inaction.

When neuritis ascends a nerve, the symptoms gradually extend in area; and if it reaches the plexus from which the nerve proceeds, they may extend to most or all the nerves of the limb. This "ascending neuritis" is not rare in man, may even reach the spinal canal, and there produce various disturbances. The inflammation may spread in the tissue outside the dura mater, or may extend to the cord, and cause subacute or chronic myelitis with or without meningitis. The paralysis that occurs secondarily to some visceral diseases, as those of the bladder, and commonly regarded as reflex paralysis, are probably produced through the agency of an ascending neuritis. Lastly, the inflammation may extend to the nerves of the other side, usually to those that correspond to the primary seat of the disease. Such extension may be through the spinal cord or the membranes, but in some cases symptoms occur in the nerve of the opposite side, without any indication of an affection of the centres. Experiments on animals have demonstrated that such implication of the opposite nerve may occur when the centres are unaffected. It has been called "sympathetic neuritis." We have seen that reflected pains are occasionally felt in the corresponding nerve on the opposite side, and it is probable that, as in neuralgia, vaso-motor disturbance in the nerve-sheath may accompany such pains, and, in a predisposed person, may cause actual neuritis.

The *Duration and Course* of neuritis vary much. A slight acute neuritis may run its course in a few weeks and then subside. Much more commonly the affection persists in a chronic stage for many weeks, or even months, and slowly passes off. The rheumatic and gouty forms are as a rule much more tedious than those that result from injury, although traumatic neuritis is sometimes very insidious and may last a long time; it may ascend the nerve from its starting-place and become localised in certain situations, where it may develop afresh, and give rise to symptoms that may seem to be independent of their cause. In the predisposed, it may set up troublesome local functional disturbance, hysterical contracture, and the like.

DIAGNOSIS.—The diagnosis of neuritis depends, first on the localisation of the symptoms to the distribution of a certain nerve-trunk, and secondly on the pain and tenderness in the nerve. The diffuse pains that attend the onset may be readily mistaken for the pains of acute rheumatism, or for those due to an inflammation of the bone, but in the course of a day or two the localisation of the symptoms declares their nature. The chronic form is easily and often mistaken for neuralgia, and the diagnosis is the more difficult because many so-called neuralgias are really due to neuritis. The distinction can only be fully discussed when we have considered the symptoms of neuralgia, but it rests chiefly on the fact that in neuralgia the pain intermits more completely than in neuritis, there is not the same initial tenderness in the nerve-trunks, and the tender spots have a more uniform localisation. Lessened sensibility or change in the muscles, showing organic damage to the nerve-fibres, are conclusive evidence of neuritis. The pains in some central diseases, chiefly those of the spinal cord, may be thought to be due to neuritis, but there is not the local tenderness of the nerve-trunk, and the pain is not limited to the distribution of a single nerve. As we shall see presently, the diagnosis of multiple neuritis from affections of the cord is far more difficult.

PROGNOSIS.—The gravest form of simple neuritis is that in which the nerve is affected secondarily to a local suppurative inflammation. The prognosis is best, as a rule, in traumatic neuritis, but the rule is one to which the exceptions are occasionally very considerable in degree. A neuropathic disposition makes the prognosis worse. In all cases the intensity of the symptoms, and the evidence of descending degeneration, furnish a more trustworthy guide to prognosis than does the mere form of the affection. The effects of complete degeneration always endure for some months. Regeneration does not occur until the original cause has ceased to act, and then occupies many weeks in its progress to the restoration of functional competence. In all forms pain is apt to linger on, and the longer the older the patient is.

TREATMENT.—The first consideration in the treatment of neuritis is the removal of its cause, if this can be discovered. A wound or injury or local inflammation adjacent to the nerve must be dealt with by appropriate measures. Any constitutional cause, such as gout, must be treated. For the inflammation of the nerve, it is of the first importance to secure to the part as perfect rest as possible. Movement causes mechanical irritation of the nerve, and involves functional stimulation of its fibres, both of which are injurious. The nerves of a limb run between muscles, and the contraction of these muscles causes pressure on the inflamed nerve, and irritates it, as the pain thus produced sufficiently shows. Pain is indeed a useful indication of the harmful influence of movement, since the stimulation of the nerves of the sheath, in which the pain is produced, doubtless has also a reflex effect, increasing the vaso-motor element in the inflammation. Hence it is most important that all movement that causes pain should be avoided. The posture of the limb should be such as to involve the least pain and the least danger of secondary contracture. The general treatment of an acute neuritis must be that suitable for any acute local inflammation, whatever its seat,—an unstimulating diet, an aperient, and diuretics. In gouty cases a brisk purgative may, with advantage, be given. General diaphoresis is useful in cases that are due to cold, and in others local sweating is often of distinct service. The limb may be steamed, or exposed to hot air. This should be followed by hot fomentations applied along the course of the affected nerve, and these by linseed-meal poultices. Leeches may also be employed at the onset of severe cases. When the inflammation results from injury, cold may be applied along the course of the nerve instead of heat; both probably modify the vascular disturbance of inflammation in a similar manner. Counter-irritation may be used at the onset of slight cases, but when the inflammation is severe, this agent is more effective during the subsidence of the inflammation than during its active stage. Blisters, repeated mustard plasters, or stimulating liniments may be used, but care must be taken not to blister skin that is anæsthetic, or troublesome ulceration may be caused. The same caution is necessary in regard to hot applications, as is shown by the case mentioned on p. 62.

Spontaneous pain requires sedatives, of which the hypodermic injection of morphia is by far the most potent. It should be used only for spontaneous pain, and not to enable the patient to use the limb in a way that would produce pain if morphia were not given. Mechanical irritation may be equally injurious, although the pain which it would cause is obviated by the sedative. Moreover it should only be used as a last resort, when the agent to be mentioned next is inadequate. Morphia has little influence on the morbid process; it lessens the effect of the disease on the brain only by acting on the brain.

In cocaine we have an agent capable to a large extent of replacing morphia, and of a greater therapeutic capacity. Although it has less power of relieving pain, it is capable of doing much more than that. It arrests the local transmission of the impulses that cause pain, and so, at a limited region, it does lessen pain and often remove it for a time, and the local character of its action enables it to exert a simultaneous influence on the morbid process. It prevents the irritative influence on the inflamed structures of the impressions that are felt as pain, which we have just considered; and the entire arrest of the influence, even though the region is small, seems to produce a much greater effect on the process than might be anticipated. It is instructive to note the opposite influence, revealed by the occasional redness of the skin and subcutaneous œdema, along the course of the inflamed nerve. The injection should be made at one of the seats of pain. In the rheumatic form salicylate of soda has been given, but is of doubtful value. Iodide of potassium sometimes seems to be useful, but no agent has so distinct an influence on the process of inflammation in the nerve as small doses of mercury. A grain of blue pill may be given once or twice a day; and if morphia has to be injected at the same time, the mercury is useful also in correcting the constipating influence of the morphia.

In the chronic stage or form, counter-irritation, by blisters or cautery, is of great value. So also is electricity, which has little influence during the acute stage. The voltaic current should be used. The positive electrode may be placed over the inflamed part of the nerve, or over the seat of pain, and kept there for ten minutes at a time, the strength of current being slight, such as the patient can just perceive. In very chronic cases a stronger current, sufficient to cause actual pain, is of service, applied in a similar manner, but for a shorter time. It has probably chiefly a counter-irritant influence, and faradism may be used in the same way. All painful impressions on the skin lessen, for a time, the nerve-pain.

The muscles supplied by the inflamed nerve may be left alone, unless their wasting is marked, or the degenerative reaction shows serious damage to the motor fibres. In cases of moderate severity they will recover when the nerve recovers. All that is desirable is that they should be gently rubbed once or twice a day. If the wasting is considerable, however, or there is degenerative reaction, they should be stimulated to gentle contraction by a weak, slowly interrupted voltaic current. They should on no account be faradised during the active stage of the affection, even if they act to faradism. The acute pain that the faradic stimulus causes, and the increased tenderness that lasts for hours afterwards, are evidence of its injurious effect.

In all cases attention to the general health is of great importance. Tonics are needed during the chronic stage. Change of air will sometimes remove, in a few weeks, symptoms that have previously been stationary for months.

The modifications of treatment that are rendered necessary by the position of the neuritis will be considered when we come to speak of the affections of the several nerves. The pathological varieties of neuritis only need special treatment in so far as they depend upon special causes, and this point, as already mentioned, must always be one of the first considerations.

MORBID GROWTHS IN NERVES: NEUROMA.

The term "neuroma" has been applied indiscriminately to all morbid growths situated on the peripheral nerves. The discovery, by Virchow, that many of these consist of a growth of nerve-fibres, while others consist of heterologous tissue such as constitutes morbid growths elsewhere, has led to the distinction of the former as "true neuromata" and the latter as "false neuromata," or "pseudo-neuromata," and even to the limitation of the term "neuroma" to a growth of nerve-tissue, whether in the central or peripheral nervous system, the heterologous growths being called by the names they bear in other situations, as "fibroma," "sarcoma," &c. The latter system of nomenclature, although certainly more consistent, has not become current in this country.

The true neuromata may consist of medullated or of non-medullated nerve-fibres, termed "myelinic" and "amyelinic" forms by Virchow. The latter were for a long time regarded as fibrous. The occurrence of ganglion-cells has been proved in only one or two instances. There is connective tissue between the nerve-fibres, which varies in amount and in character, and hence the firmness of these tumours also varies. This interstitial tissue may be so abundant as to constitute an intermediate form between the true and false varieties. This is probably the condition in most cases of multiple neuroma. The "false neuromata" may be of various nature, but fibrous growths, "fibromata," are far more common than any other kind. Myxoma occasionally occurs, the new mucoid tissue growing from the nerve-sheath. Glioma is very rare on the peripheral nerves, although occasionally found on the auditory nerve. Various forms of sarcoma have been met with. Carcinoma also occurs, very rarely as an isolated growth, but not uncommonly as a more or less diffused or nodular infiltration of the nerve, arising by extension from a contiguous growth. Syphilitic growths are common on the cranial nerves within the skull, but are rare elsewhere. In lepra anæsthetica the nerves are infiltrated with fibrous tissue, enlarging them to many times their normal size. The enlargement is rarely nodular, and is rather a chronic cirrhotic inflammation than a growth. (See "Leprous Neuritis.")

A curious variety of neuroma, consisting of interlacing cords, more or less nodular and tortuous, is termed *plexiform neuroma.* About twenty cases have been recorded. The disease commonly begins in fœtal life, and is most common on some branch of the fifth nerve in the orbit or the upper eyelid, but has been met with over the temporal bone, in the lumbar, cervical, brachial, and solar plexuses, on the penis and the mamma. It may be quite superficial or deeply seated. The cords, from 1 mm. to several centimetres in diameter, consist of a clear outer zone of concentric fibrillar connective tissue, of inner looser nucleated tissue, and, in the middle, a bundle of nerve-fibres, some normal, others degenerating. The cords are connected together by loose tissue, sometimes myxomatous, separated from the proper tissue of the cords by an epitheliated space. The growth of this form is extremely slow, but it may exert compression on adjacent structures.* A remarkable case of extensive myxomatous disease of the nerves of the forearm, congenital in origin, presenting analogies to plexiform neuroma, is recorded by Mr. De Morgan.†

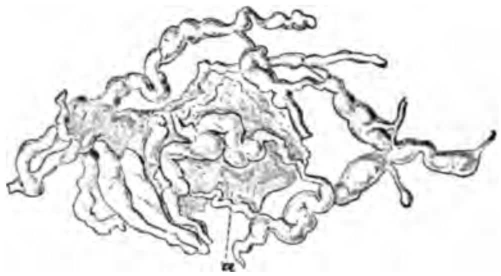

FIG. 45.—Plexiform neuroma from the orbit. (After Marchand.) The connective tissue surrounding the cords has been removed except at *a*, and irregular, cylindrical, nodular cords are seen anastomosing. A nerve passes into the tumour and suddenly becomes enlarged.

The subcutaneous extremities of sensory nerves are sometimes enlarged into minute tumours which, when painful, have been termed "tubercula dolorosa." Neuromata in the skin may co-exist with similar tumours on the nerve-trunks. Those shown in Fig. 46 are from the same case as the tumours represented in Figs. 47—49.

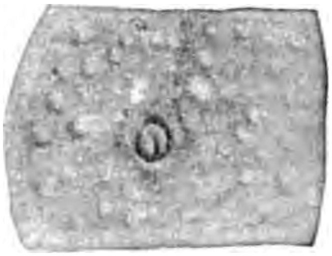

FIG. 46.—Cutaneous neuromata from skin of abdomen. (After Smith.)

The size attained by growths on nerves varies from that of a child's head to a nodule only just visible. They rarely exceed the dimensions of the closed fist. The variation in size of multiple neuromata is illustrated in Figs. 47—49, after Smith.‡ The large tumour (Fig. 47) was the size of a lemon, but in the same case a growth from the sciatic plexus almost filled the pelvis. They may occur on any nerve in the body, cranial or spinal, and are

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* Marchand, 'Virchow's Archiv,' Bd. lxxvi, p. 36.

† 'Path. Trans.,' vol. xxvi, p. 2.

‡ R. W. Smith, 'Treatise on Neuroma,' 1849. Many instructive cases have been collected by Bowlby ('Diseases and Injuries of Nerves').

FIG. 47.

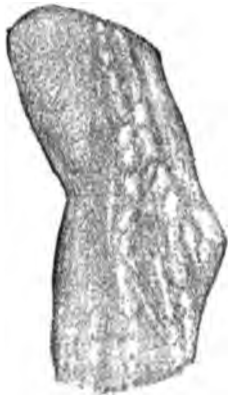

FIG. 48.

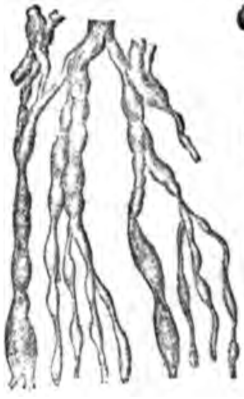

FIG. 49.

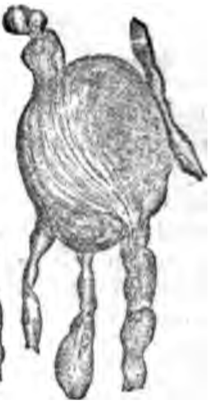

FIG. 50.

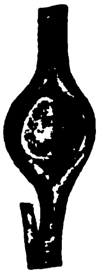

FIG. 47.—Right elbow of a man, æt. 30, with multiple neuromata; those on the nerves of the arm are visible beneath the skin as moniliform series of swellings along the course of the nerves. (After Smith.)

FIG. 48.—Part of the brachial plexus and nerves of the arm.

FIG. 49.—Neuroma of the posterior tibial nerve, same case. The tumour was the size of a lemon, and could be felt in the popliteal space. The flattened bundles of fibres of the nerve were separated and passed over the surface of the tumour. The portion of nerve to the right is a piece of the external popliteal. The tumours caused no interference with function. The patient died of enteric fever. One large tumour filled the pelvis.

FIG. 50.—Small neuroma of popliteal nerve laid open. It is seen to lie within the sheath. It caused no symptoms during life. (After Smith.)

sometimes numerous on the pneumogastrics, spinal roots, and sympathetic plexuses. When neuromata are multiple they are often extremely numerous, and almost every nerve in the body may be transformed into a chain of growths. In the case figured more than 200 were counted in the right arm alone, and the total number of growths in the body cannot have been less than 1000, while in another 3020 were counted.*

The tumours are almost always within the sheath of the nerve (Fig. 45). Sometimes they are on one side, and the nerve may pass by unchanged. More often the substance of the nerve is involved, and the fibres may be separated and spread out on the surface (Fig. 44). Even then they may not be damaged. They suffer far more in heterologous growths than in true neuromata.

CAUSES.—The causes of neuroma are generally obscure. Multiple neuromata are sometimes hereditary, and are probably due, in most cases, to a congenital tendency of tissue growth. Plexiform and multiple neuromata have been met with in the same family. They are said to be sometimes the result of general neurotic predisposition, and evidence of this is found in the occasional occurrence of neuromata in the subjects of cretinism or idiocy, of which some remarkable examples have been recorded. Multiple neuromata are said to be almost confined to men. The isolated form is not uncommon in women.

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* 'Med. Times and Gaz.,' 1883, i, p. 152.

They may occur at any age. Virchow believes that they are unusually common in the phthisical and scrofulous.

Of traceable immediate causes the most frequent is traumatic injury. Pressure, punctured wounds, or division of the nerves may give rise to them, apparently by a perversion in the process by which cicatrisation takes place. A well-known instance is the formation of the so-called "amputation neuromata," or "bulbous nerves," round or oval growths, the size of a bean or larger, which form on the divided extremities of the nerves in the stump left after an amputation.

The occurrence of new growths containing nerve-fibres may seem to be very remarkable. We must remember, however,. how frequently they are connected with traumatic causes, *i. e.* take origin in cicatricial processes. In such processes the nerve-fibres present a remarkable power of growth. Ranvier has shown that from the end of each old fibre several new fibres grow, only one of which probably ultimately persists. Under apparently mechanical influences some of these fibres may twist about, and even turn and grow upwards. It is thus not difficult to understand that a true neuroma may result from injury to a nerve; it is said, indeed, that the ends of the nerves in amputation stumps are generally enlarged into minute bulbs, which contain more fibres than do the nerves on which they are situated.*

SYMPTOMS.—These may be entirely absent, especially in the case of multiple (true) neuromata, which are sometimes discovered post mortem, when their existence has not been suspected. Often, however, they cause marked symptoms. The most frequent is pain seated in the tumour (which may be extremely tender) or referred to the distribution of the nerve, often acute, burning, or darting, and frequently intermittent. It may be increased by pressure on the tumour. In the case of the terminal neuromata local pain may be the only sensory symptom; but when the nerve-fibres suffer, paræsthesiæ, numbness, or formication may be complained of, and ultimately sensation may be lessened or lost. Paralysis of the muscles supplied by the nerve is occasionally met with, the region paralysed corresponding to the distribution of the nerve on which the tumour is situated, except in the case of neuromata in the cauda equina, from which paraplegia may result, with flexor spasm. A neuroma of the pneumogastric may cause grave cardiac disturbance, sometimes increased by pressure on the tumour.

More common motor symptoms are reflex spasms in adjacent or even distant muscles. For instance, in a case of "bulbous nerves" in the stump after amputation at the shoulder-joint, the muscles on that side of the neck were in constant clonic spasm. Occasionally epileptiform convulsions are produced, and have been known to cease when a painful neuroma had been excised. The occurrence of these symptoms has little relation to the size of the tumour.

When the neuromata are in accessible situations they may be felt. Sometimes they give rise to visible tumours, and in multiple neuroma the course of the superficial nerves may be marked out by lines of bead-like swellings, as in Fig. 47. The cutaneous neuromata also cause visible swellings. The whole abdomen was covered with such nodules in the case recorded by Smith (Fig. 46). They are sometimes not sensitive, sometimes exquisitely tender and most painful, especially when closely united with the skin. Pressure on the nerve above the tumour commonly lessens the pain.

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* Bowlby, loc. cit., p. 20.

Plexiform neuroma rarely causes other symptoms than the signs manifesting its local presence. The functions of the nerve-fibres involved are not usually interfered with. Once or twice anæsthesia of the skin has been observed.

Course.—The growth of neuromata varies greatly in rapidity. When symptoms occur they most commonly last for a long time, increasing in severity, and from the long-continued pain the patient may ultimately be worn out. In other cases the symptoms show little tendency to increase, and in rare instances they may lessen in severity and even disappear.

Diagnosis.—The recognition of the existence of a neuroma depends on its superficial position, or on the production of symptoms of damage to the nerve. The latter, when existing alone, are equivocal, since they may be due to pressure on the nerve from an adjacent growth, or even to neuritis. The disease may be suspected if symptoms—pain, numbness, weakness—occur, limited to the distribution of a nerve-trunk, and no cause of external pressure can be discovered, and the long course of the symptoms makes it unlikely that they are due to neuritis. The diagnosis can, however, only be certainly made when the tumour can be felt. Secondary subcutaneous sarcomata may very closely resemble neuromata.

The diagnosis of the kind of neuroma, whether "true" or "false," *i. e.* composed of nerve-fibres or other tissue, is very difficult. Multiple neuromata are usually true, and of very slow growth; false neuromata are rarely multiple unless they are of infecting nature and rapid in development. The existence of idiocy, or other neuropathic indication, is in favour of the neural nature of the tumours. The lateral position of the growth on the nerve is in favour of its "false" character. The diagnosis of plexiform neuroma is only possible when the tortuous nodular cords can be felt.

Prognosis.—This depends on the existence of symptoms. If these are absent, unless the tumour is of rapid growth, there is good reason to hope that they will not occur. In multiple (true) neuromata the nerve is less likely to suffer than in isolated growths. If symptoms are present the prognosis is less favorable, and depends on the tendency which they exhibit to increase.

Treatment.—Medicines are useless except in the syphilitic forms of nerve-tumour, not here considered. Extirpation is the only remedy. If the tumour is lateral, and can be shelled out, the removal entails little risk. If, however, the growth infiltrates the substance of the nerve, the affected part must be excised, and the ends brought together. The risk of permanent loss of function in the nerve is great, and in deciding on such an operation, the urgency of the symptoms, and the importance of the function of the nerve, must be taken into consideration. After the extirpation of such tumours, the tendency of cicatricial processes in nerves to go on to the formation of similar growths (seen in " bulbous nerves ") renders the chance of relapse considerable. The operation reproduces conditions favorable for the activity of whatever predisposing influence aided in the original development of the disease. When neuromata are multiple, surgical treatment is out of the question, except for isolated tumours that cause distress.

In the case of terminal neuromata—whether the cutaneous " tubercula dolorosa " or the amputation neuromata—excision is practicable, and it is unattended with the risks attending interference with a nerve in its continuity. In amputation flaps the chance of a relapse is considerable.

In plexiform neuroma excision is undesirable, and no other treatment is of any use.

Pain, and the reflex spasm, that are so distressing in amputation neuromata, need relief when attempts to cure have failed. By nerve-stretching we may perhaps give relief without the effects that follow excision, and in cocaine we have a means of arresting for a time the ingoing impulses that play an important part in generating the symptoms. The rest thus given to the centre is absolute, and its degree and frequent repetition may compensate for its brevity.

DISEASES OF SPECIAL NERVES.

The diseases of the cranial nerves may be most conveniently considered in connection with the diseases of the brain. Here, therefore, only the affections of the spinal nerves will be described, in so far as they present special features. The effect of paralysis of the individual muscles has been already described in detail, and need not be here repeated, except in general terms, or in so far as the association of palsies resulting from a nerve-lesion presents a particular character.

Phrenic Nerve.—Impaired function of the phrenic nerve is commonly the result of disease of the spinal cord, or of the roots of the nerve from disease of the membranes or the bones. The deep position of the nerve-trunk protects it from injury, but it is occasionally damaged in wounds of the neck, and in its course through the thorax it may be compressed by tumours, aneurisms, &c. Paralysis sometimes follows exposure to cold, and is ascribed to neuritis. This has been met with in multiple neuritis. In disease of the spinal cord and membranes both nerves are usually paralysed; causes acting on the nerve-trunk usually affect one only. The effect and symptom of paralysis is inaction of the diaphragm (see p. 22). If one nerve only is affected, the diaphragm does not descend on that side, but the movement of the other side lessens the resulting defect of movement, and it can then be detected only by close observation. The loss of the action of the diaphragm has little effect on the respiratory functions while the patient is at rest, but dyspnœa is said to be readily produced by exertion; the breathing then becomes quick and the voice feeble. At the same time this is probably chiefly because too much and too constant work is thrown on the upper thoracic muscle of extraordinary breathing. Any lung disease, such as an attack of bronchitis, is rendered far more serious by the diminished breathing power. When the diaphragm is paralysed, the movement of the thorax is often increased, and the expansion of the lower part may draw forwards the adjacent abdominal wall. This must not be mistaken for the effect of the descent of the diaphragm. Paralysis has to be distinguished from (1) abnormal nervous breathing. The diaphragm is used little in extraordinary breathing, which is chiefly by the upper part of the thorax, of the "superior costal type," as it is called. Hysterical and nervous patients will often breathe, for a time, only in this manner, even when at perfect rest, especially when they are under observation. Such breathing is no doubt facilitated in women by the fact that the diaphragm is habitually used by them less than by men. Repeated examination may be necessary to determine whether there is any real paralysis of the diaphragm in these cases. A single diaphragmatic inspiration settles the point. The patient's attention should be distracted, and she should not be aware of the object of the examination. (2) Inflammation of the diaphragm may arrest its movement, and so also may diaphragmatic pleurisy or peritonitis—each distinguished by the fact that it is usually secondary to adjacent inflammation, and any movement, if effected, causes characteristic pain. (3) A primary and isolated degeneration of the muscular fibres of the diaphragm has been described by Callender and others as common after death, but it has not yet been proved to cause such inaction, during life, as might be confounded with paralysis.

Paralysis may be due to disease of the nerve or of the spinal cord. In the latter case other muscles always suffer; in the former the diaphragm usually suffers alone unless it is part of multiple neuritis. In disease of the nerve-roots there are other indications of the position of the disease. The affection is often overlooked because not searched for, especially in disease of the spinal cord. The observer forgets that its associations are not with the lower dorsal muscles, and he omits to look for it in cases in which the cervical cord is suffering and other respiratory muscles act well.

If there is reason to suspect neuritis, counter-irritation should be applied over the lower and inner part of the anterior triangle of the neck. The only other special point in treatment is the application of electricity. The nerve may be stimulated by pressing the rheophore deeply outside the lower part of the clavicular portion of the sterno-mastoid. The other pole may be placed at the epigastrium or over the corresponding half of the diaphragm. But the influence of electricity on paralysis of the diaphragm is not sufficient to make its use desirable. In the cases in which such treatment could do good the affection is transient and comparatively unimportant. In central disease electricity has very little influence.

NERVES OF THE UPPER LIMB.

The nerves of the arm and shoulder are derived from the five lower cervical and the first dorsal nerves. These interlace in the brachial plexus in such a complex manner that most of the nerves of the arm are derived from many spinal roots.

The nerve-roots form, by their union, three trunks, which we may indicate by Roman numerals. They are formed thus :—I, by the branch from the fourth, and the roots of the fifth and sixth cervical; II, by the seventh; and III, by the eighth cervical and the first dorsal. Each trunk divides into two parts, and the union of these divisions forms the three cords of the brachial plexus from which the nerves of the arm proceed. But before the primary trunks divide, certain nerves arise, the origin of which is thus less doubtful. The fifth and sixth cervical roots give origin directly to the posterior thoracic nerve for the serratus, and from the cord formed by their union springs the supra-scapular nerve. The three cords of the plexus have the following relations :—The posterior is derived from all three primary trunks, and gives rise to the subscapular nerve, the circumflex, and the musculo-spiral (or radial, as the whole nerve is sometimes termed). The upper or outer cord is derived from the two upper primary trunks, *i. e.* from the fourth, fifth, sixth, and seventh cervical roots, and from it proceed one anterior thoracic and the musculo-cutaneous nerves, together with the outer head of the median. The inner or lower cord is derived only from the lowest primary trunk, *i. e.* from the last cervical and first dorsal, and gives rise to the ulnar, the inner head of the median, the internal cutaneous, the intercosto-humeral, and to the second anterior thoracic nerves. It may be convenient to put these coarse anatomical relations in the form of a table.

Nerves.		*Primary Trunks.*		*Nerves.*
Subscapular . Circumflex . Musculo-spiral	Post. cord	I. 4, 5, and 6 C.	Upper cord	External ant. thoracic. Musculo-cutaneous. Outer head of median.
		II. 6 and 7 C.		
		III. 8 C. 1 D.	Lower cord	Inner head of median. Ulnar. Internal cutaneous. Intercosto-humeral. Internal ant. thoracic.

These anatomical facts, however, give us little help in tracing the relation of the nerves to the spinal roots. The investigations of Ferrier and Yeo,*, who ascertained the movements produced by faradising the several spinal roots in the monkey, show us the way in which the movements, muscles, and nerves are represented in the spinal roots. Their results are therefore of great interest, although we are not justified in transferring the facts to man except in so far as they receive confirmation from human anatomy and pathology.† Subject to this reservation, the chief results are important, and may be thus stated.‡

The roots to which the several nerves are thus traced are as follows: Subscapular, 6 and 7 C.; circumflex, 4 and 5 C.; musculo-spiral, 4, 5, 6, 7, 8 C.; musculo-cutaneous, 4, 5 C.; median, 5, 6, 7, 8 C.; ulnar, 8 C., 1 D.

Still more important are their observations on the relation of various movements of the arm to the nerve-roots, and these are as follows:

Cervical 4.—Elevation and retraction of the arm, flexion and supination of the forearm; by the rhomboids, supra- and infra-spinatus, biceps, brachialis, and supinators.

Cervical 5.—Similar to the last, but without retraction of the arm, and with extension of the wrist and first phalanges; by the deltoid, serratus, flexors of elbow, extensors of wrist, and long extensors of fingers.

Cervical 6.—Adduction and retraction of the upper arm, extension and pronation of the forearm, flexion of the wrist; by contraction of the pectoralis, latissimus dorsi, triceps, flexors of wrist, pronators.

Cervical 7.—Adduction and rotation inwards at the shoulder-joint, flexion of the wrist, and of the fingers at the second phalanx; by the teres major, latissimus dorsi, subscapularis, triceps, and long flexors of the fingers.

Cervical 8.—Flexion of fingers and thumb so as to close the fist; flexion of wrist towards the ulnar side, pronation of forearm, extension of elbow; by the intrinsic muscles of the hand, the long flexors of the fingers and thumb, the flexors of the wrist, and the triceps.

Dorsal 1.—Adduction of the thumb, flexion of the fingers at the metacarpo-phalangeal joints; by the interossei, &c.

Thus most movements are related to many spinal roots. The most important relations are these: that of the deltoid, rhomboids, supra- and infra-spinatus, flexors of the elbow, and supinators, to the fourth and fifth cervical; that of the adductors of the arm and extensors of the elbow to the sixth and seventh nerves; pronation to the sixth and eighth; extension of the wrist to the fifth; flexion to the eighth; extension of the first phalanx to the fifth; flexion of the fingers to the seventh and eighth, and the action of the intrinsic muscles of the hand to the first dorsal. These facts will probably, in the future, find important practical applications.

Regarding the relation of the sensory branches to the nerve-roots, we have besides the facts of anatomy, only the evidence furnished by rare cases of injury or disease in which the lesion and its effects are well-defined.* Few facts of this character are, however, so conclusive as that illustrated in Fig. 51, in which the area shaded by slanting lines was rendered anæsthetic by the division of the posterior roots of the last cervical and first dorsal nerves. The general indications are that the distribution of the sensory fibres of the nerve-roots is along the axis of the limb and not across it. The last two pairs of nerves supply the ulnar side of the limb and the finger tips, the fifth pair the radial side and the middle roots of the brachial plexus, the middle zone of each side of the limb and the thumb.†

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* 'Proc. Roy. Soc.,' March 21, 1881. Careful dissections by Herringham (cf. March 25th, 1887) give somewhat different results, but this method of investigation is open to more uncertainties than that of stimulation. Herringham's conclusions should, however, be noted by future investigators.

† In the case of the leg there are some important discrepancies between the results in the ape and the conditions that obtain in man. These will be noticed in their proper place.

‡ Ferrier has since stated that the relations he gave (followed in the text) were all one nerve too high ('Proc. Roy. Soc.,' 1883, vol. xxxv, p. 229), but this would make the innervation of the intrinsic muscles of the hand from the second dorsal nerve, which is certainly not the case in man.

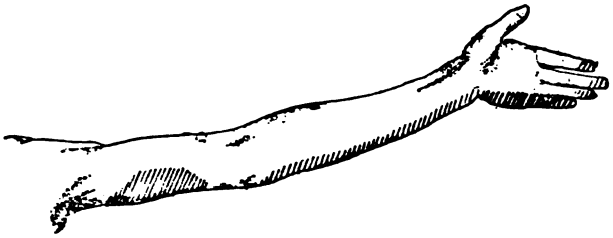

FIG. 51.—Area of impaired sensation after division of the posterior branches of the 8th C. and 1 D. nerves, by Professor Horsley for inveterate paroxysmal neuralgia in that region.

Paralysis of the nerves of the upper limb may be due to disease of the nerve-roots, of the plexus, or of the trunks that arise from these. It is convenient to consider, first, the diseases of the nerve-trunks, and afterwards those of the nerve-roots and of the plexus. From the roots themselves only one important nerve-trunk is derived: the posterior thoracic nerve. The others spring from the plexus.

The morbid influences that affect the nerves of the arm are very varied. The brachial plexus passes close to the shoulder-joint, and hence dislocation often damages the nerves, sometimes one only, sometimes several, and occasionally all the trunks. In fractures of the bones, the nerves adjacent may be torn, or may be compressed by the ends of the bone, or by the callus that is formed, or by bandages that are applied. The course of certain nerves is, in some parts of the arm, especially apt to be the seat of wounds of various kinds. They may be compressed by cicatricial fibrous tissue when the wound is merely near the nerves, while the position of some exposes them to pressure against the unyielding bone. Even slight pressure on these nerves is effective when prolonged, as it often is during sleep, when warning sensations are unperceived. The sleep induced by alcohol is especially apt to be attended by this accident. Hence there is a group of "*Sleep-palsies*" of the arm, chiefly of the musculo-spiral and median nerves, rarely of the plexus itself.

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* Instructive facts of this character will be found in papers by Thorburn, 'Brain,' January, 1887, and October, 1887.

† This is due to the mode of growth of the arm-bud in the embryo; as shown by Ross in a most instructive paper, 'Brain,' January 7th, 1888. See also chapter on the Functions of the Spinal Cord.

Primary neuritis may involve any nerve, or the brachial plexus, or the nerve-roots; certain nerves also suffer in multiple neuritis. Secondary neuritis, set up by injury or by adjacent inflammation, may extend upwards, and, reaching the plexus, spread to other nerves. Neuromata are rare. Various morbid processes in the neck may involve the nerve-roots. Neuritis of the plexus, and the multiple form, are separately described.

Posterior Thoracic Nerve—Nerve to the Serratus.—In consequence of its position and long course, the nerve to the serratus often suffers. After being formed in the substance of the scalenus medius muscle, by the union of branches of the fifth and sixth cervical nerves, it passes behind the brachial plexus and along the side of the chest to the lower border of its muscle. It is in the neck that the nerve is most often injured. It may be damaged there by direct pressure—as in carrying on the shoulder a heavy sharp-cornered object—or by severe muscular efforts, such as carrying a weight that does not press into the neck, wielding a heavy hammer, or long-continued exertion with the raised arm, as, for instance, in whitewashing a ceiling. In such cases a traumatic neuritis is set up by the violent compression of the nerve consequent on the forcible or prolonged contraction of the muscle through which it passes. When the onset of the palsy occurs immediately on the exertion, a paralysing injury to the nerve must have occurred, which will be followed by secondary neuritis. Falls and blows on the neck and shoulder are occasional causes; there is usually much bruising of the adjacent parts. Punctured and gunshot wounds occasionally involve the nerve. The least frequent cause is exposure to cold, such as a draught on the neck, or sleeping on damp earth. I have once known it to follow parturition, developing four days afterwards, doubtless from neuritis excited either by the muscular exertion, or by exposure of the neck to cold during the labour. The serratus suffers also in central disease, chiefly in progressive muscular atrophy and infantile paralysis; but in these it is always associated with palsy of other muscles. Isolated paralysis of this nerve is nine times as frequent in men as in women. It occurs chiefly in muscular workers, and during the active period of life, twenty-five to forty. The immediate causes sufficiently explain this relation to age and sex, and they also account for another fact—that it is far more frequent on the right side than on the left. It is sometimes bilateral, but the two nerves are never damaged at the same time. One man was affected on the right side after carrying heavy beams on the shoulder; he then carried them on the left shoulder, and the left serratus became paralysed.

Severe neuralgic pains in the neck and about the shoulder commonly precede and accompany the onset of the affection when due to neuritis. The symptoms of the paralysis of the serratus have been already described (p. 29). It is readily recognised by the recession of the posterior edge of the scapula from the thorax when the arm is put forwards

(Fig. 8, p. 29). In severe cases the muscle is found to have lost faradic irritability, although it may still contract to the voltaic current.

The course of a severe case is always tedious. Months may pass before improvement is manifested, and the paralysis is sometimes permanent. In treatment it is desirable to maintain the nutrition of the muscle, as far as possible, by electrical stimulation, and its superficial situation renders this easy. If counter-irritation is desirable, it should be applied over the position of the scalenus, since the lesion of the nerve is commonly at this place. The use of the arm should be limited. When practicable, the elbow should be carried in a sling of such a length as slightly to raise the shoulder. All movements that may involve contraction of the scalenus, *i.e.* all movements needing elevation of the shoulder, must be prohibited.

The *Supra-scapular nerve* arises from the trunk formed by the union of the sixth, fifth, and a branch of the fourth cervical nerves, but its fibres come from the fifth and partly from the fourth. It is occasionally damaged alone, or with the circumflex, in dislocation of the humerus, and by falls on the shoulder, and by carrying heavy weights. In some cases the injury may be to the upper part of the plexus (see p. 91), which may recover except this nerve. The result of its disease is palsy of the supra- and infra-spinatus (p. 30). The latter is the more important, and causes a defect of the rotation outwards of the humerus, interfering with many movements, and, among others, with the movement of the pen along the line in writing. Undue work is thrown on the posterior part of the deltoid and the teres minor; the deltoid may become hypertrophied, and its posterior edge is conspicuous against the wasted infra-spinatus. The scapula is rotated, so that the lower angle is moved upwards and inwards. The paralysis of the supra-spinatus is unimportant, except that the humerus is less firmly fixed and the deltoid more readily fatigued. It can, in very slight degree, supplement the deltoid, and if paralysed with the deltoid, the head of the humerus falls more than it does when the deltoid is paralysed alone. Some anæsthesia over the scapula often results from disease of this nerve.

The *Circumflex nerve*, although arising from the posterior cord of the plexus, seems to derive its fibres from the same source as the supra-scapular, the fourth and fifth cervical nerves. It supplies the deltoid and teres minor, and the skin over the deltoid. The course of the nerve renders it very liable to injury from dislocations and falls on the shoulder, and from the pressure of a crutch. Simple neuritis and so-called "rheumatic palsy" are rare, but the nerve has been affected in some acute diseases, possibly from pressure, and in rheumatic fever, perhaps by inflammation extending from the joint. It suffers, with other nerves, from disease of the upper part of the brachial plexus.

The chief symptom is paralysis of the deltoid (p. 29), which abolishes almost all power of raising the arm, a very trifling degree of abduction by the supra-supinatus alone remaining. The slight nerve-supply that the fore-part of the deltoid receives from one of the anterior thoracic nerves is insufficient to mitigate the effect of the paralysis of the circumflex, although it may maintain slight power of voluntary contraction in the anterior fibres, and this sometimes gives rise to an erroneous impression that the paralysis of the circumflex nerve is incomplete. The effect of the conjoined palsy of the teres minor is unimportant. The wasting of the deltoid causes a change in the shape of the shoulder (Figs. 7 and 9). Sensation may be lost in the skin over the lower part of the muscle, and the loss sometimes extends on to the shoulder (Fig. 53). In some cases there is no anæsthesia, even when the muscle is wholly paralysed; we have seen that this is often the case in nerve-lesions (p. 60). Adhesions are apt to form in the shoulder-joint, in part probably the result of trophic changes, since the circumflex supplies the articulation as well as the chief muscle that moves it.

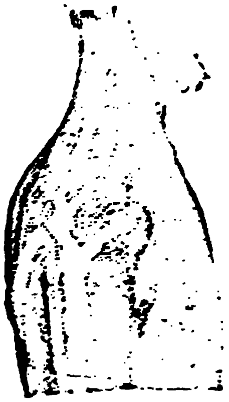

FIG. 52.—Disease of the circumflex nerve from pressure-neuritis during acute illness; wasting of deltoid. The dotted line indicates the area of cutaneous anæsthesia.

The diagnosis of the paralysis of the circumflex nerve is easy. The only condition readily mistaken for it is anchylosis of the shoulder-joint in a stout individual, in whom the state of nutrition of the muscle is not readily perceived. The risk of error is increased by the fact that arthritis and paralysis may result from the same cause—a fall on the shoulder. Passive movement at once solves the problem. The scapula moves with the arm in anchylosis, and not in palsy. The importance of examining the joint is very great in all cases of interference with movement of the shoulder. A patient had an epileptic fit which was supposed to have left brachial monoplegia, merely because he fell on the shoulder, and the movement of the arm was lessened first by pain, and then by arthritic adhesions.

The *Musculo-cutaneous nerve* supplies the chief flexors of the elbow and the skin over the radial side of the forearm. It is scarcely ever paralysed alone, but often suffers, with other nerves, in disease of the brachial plexus. The symptoms correspond to its function; there is paralysis of the biceps and brachialis (see pp. 32, 33), the effect of which is especially conspicuous when the arm is supinated and the supinator longus cannot flex the elbow. There may also be anæsthesia of the radial side of the forearm, front and back.

The *Musculo-spiral nerve* is more frequently paralysed alone than any other nerve of the arm. Arising from the posterior cord of the brachial plexus, it seems to derive its motor fibres from all the nerve-roots that enter the plexus except the first dorsal. It supplies the triceps, all the muscles on the back of the forearm, the extensors of the wrist and fingers, both the supinators, also the skin on the radial side of the back of the hand, the back of the thumb, index finger, and half the middle finger. It is thus the extensor nerve of the arm, and has a more complete relation to a single function than is common among the nerves of the limbs; a fact that has given rise to diagnostic error, since a palsy limited to a single function suggests a central cause. The frequency with which this nerve suffers is due to its course. As it leaves the brachial plexus to wind round the bone, its position exposes it to injury in dislocation or from the pressure of a crutch; the most common form of "crutch palsy" is paralysis of this nerve. Lying as it does close to the humerus, it is readily torn in fracture, or compressed by callus, and it suffers gravely from even temporary pressure against the hard bone. Such pressure is often exerted during sleep. The nerve may be paralysed by the pressure to which it is exposed when the body, with the arm beneath it, rests on the ground or on a hard bed. Thus, a man slept all night on a bench, lying on his right side with his arm beneath him, and woke in the morning with this nerve paralysed. Curiously, a precisely similar event had occurred to him three years before. In other cases the pressure on the nerve is that of a hard and sharp object over which the arm is placed during sleep, as the edge of a chair or the side of a couch. The Continental custom of tying together, behind the body, the arms of a prisoner, often causes paralysis of this nerve, sometimes on both sides. In Russia it is not uncommon in infants, from the popular practice of binding the arms to the body and then laying the child to sleep on its side. I have three times seen paralysis from a violent contraction of the triceps, once during the act of pulling on a tight pair of boots, once from throwing a stone with energy, and once from grasping a lamp-post to avoid a fall during a severe attack of giddiness. In each the nerve was at once completely paralysed; and in the second, in which the palsy was severe, a bruised appearance was observed over the lower part of the triceps. Neuritis due to cold is often assumed to be a cause, and is possibly sometimes effective, but the exposure has usually been during alcoholic sleep, and it is probable that the paralysis is due to compression rather than to cold. The same influence may also have been operative in cases in which the nerve has been paralysed during an acute disease. Although in a case of paralysis during typhus, described by Bernhardt, neuritis was found after death, this was at the spot at which pressure is usually effective, and the inflammation was probably thus excited. Pressure always sets up neuritis, and the diagnosis of a primary neuritis is never justified if the spot affected is one liable to compression.

The symptoms of disease of the musculo-spiral nerve are paralysis of the extensors of the elbow and of the wrist, the long extensors of the fingers and thumb, and the supinators (see pp. 33, 34). All these are paralysed by a complete lesion of the nerve near the brachial plexus. When the lesion is near the middle of the humerus, as it is in most cases of compression, the triceps generally escapes, but not always. The supinator longus is usually paralysed, but escapes if the lesion is below the origin of its branch, and may also escape if the injury to the nerve is incomplete. In sleep palsy it usually suffers, but I have seen it unaffected, in a case in which the ext. carpi radialis was also but little affected, and Bernhardt has noted the escape of the supinator in an otherwise complete paralysis from dislocation of the humerus. The extensor palsy causes characteristic wrist-drop, and loss of the power of extending the first phalanges of the fingers and the thumb (see p. 34). There is sometimes a gradation of palsy from the first finger, in which it is least, to the fourth, in which it is greatest (Fig. 54). The same gradation is seen in lead palsy, &c. It is not easy to explain. The action of the flexors is feeble, from the loss of antergic support; in most cases of complete palsy I have found the power of flexion reduced from this cause to one third of the normal. The loss of the power of supination is a grave inconvenience. If an object is grasped firmly the arm becomes pronated. The patient tries to compensate for the loss by putting the elbow against the side and rotating the humerus. The pronators may ultimately become shortened. The over-flexion of the carpus, and its deficient support by the extensor tendons, leads to a prominence of synovial sacs, and perhaps of the bones, at the back of the carpus (Fig. 55). In severe cases the muscles waste, and the maximum circumference of the limb below the elbow may be a quarter or half an inch less than on the other side. The electrical reactions depend on the severity of the lesion of the nerve; commonly there is well-marked degene-

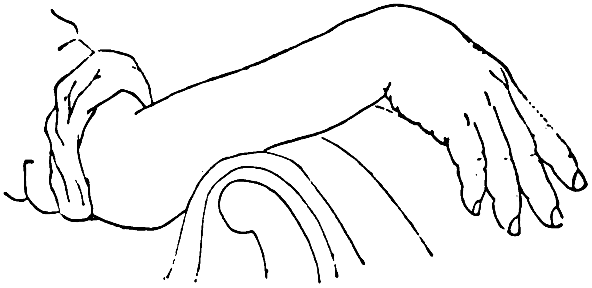

FIG. 54.—Paralysis of the musculo-spiral nerve; maximum extension of wrist and fingers. The extension of the fingers progressively diminishes from the first to the fourth. (From a photograph.)

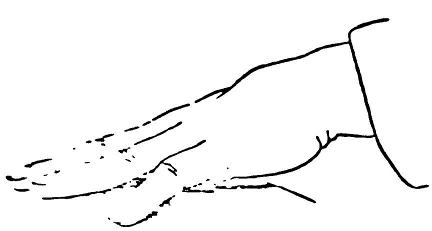

FIG. 55.—Prominence at back of hand from paralysis of the extensors. The patient was suffering from wrist-drop due to silver-poisoning (see vol. i., "Argyria.")

rative reaction. The affection of sensibility in the area supplied by the nerve is very variable. The skin of the upper arm rarely loses feeling; in the hand sensation may be normal, although the muscular paralysis is complete. There may be subjective "tingling" in the part although there is no loss of sensibility.

The diagnosis is easy in most cases. The affection is distinguished from lead palsy by its common limitation to one arm, by the affection of the supinator, and often by the sudden onset; that from lead is gradual. These characters, and the commonly obtrusive cause, always suffice for the diagnosis. It must be remembered, however, that this nerve is one most frequently affected in many other forms of multiple neuritis (*q. v.*). The prognosis in disease of the musculo-spiral nerve depends on the severity of the lesion, as indicated by the electrical reaction. When there is evidence of nerve-degeneration the paralysis usually lasts for some months. Recovery ultimately occurs in almost all cases. The treatment is that for neuritis, already described, but attention should be paid to the posture of the limb, so as to avoid tension on the affected part of the nerve. For this reason, and also because strong contraction of the triceps may further injure the nerve, extension of the elbow should be avoided in cases of lesion of the nerve as it winds round the humerus.

The *Median nerve* supplies the pronators, the radial flexor of the wrist, the flexors of the fingers (except the ulnar half of the deep flexor), the muscles that abduct and flex the thumb, and the two radial lumbricales. Its motor fibres seem to have an extensive origin from all the cervical roots that enter the brachial plexus. It subserves sensation on the radial side of the palm, on the front of the thumb, of the first two fingers, and of half the third finger, and also, at least in many persons, on the back of the last phalanx of the index and middle finger; sometimes also on the adjacent part of the back of the ring finger and the back of the last phalanx of the thumb. Isolated palsy of this nerve is not frequent, and generally results from wounds of the forearm, or fractures of the forearm bones, rarely from injuries in the upper arm. The nerve is occasionally the seat of neuritis, which may be induced by compression, especially in those who, in their work, have to hold an object between the hand and upper arm. I have known it to be paralysed at the wrist a few hours after a severe sprain of this joint. Webber has recorded a case in which it seemed to be injured by a violent contraction of the pronator teres. When there is complete damage to the nerve, pronation is impossible beyond the mid-position, to which the supinator longus can bring the forearm, and an attempt is made to supplement this by rotating the humerus inwards, and separating the elbow from the side. The wrist can only be flexed with a strong inclination towards the ulnar side. The thumb is in persistent extension and adduction (like the thumb of the ape, Figs. 22 and 23, p. 38), and cannot be opposed to the tips of the fingers. The power of flexing the second phalanges on the first is lost, and also that of flexing the distal phalanx of the first and second fingers, but this phalanx can still be flexed in the third and fourth fingers, by the ulnar half of the flexor profundus (see p. 29). Interosseal flexion of the first phalanx is still possible, and the unopposed extensor action of the interossei on the middle and distal phalanges tends to cause a subluxation of the articulations concerned. The wasting of the thenar muscles is usually conspicuous. The resulting condition is very characteristic, but if the damage is at the wrist flexion of the wrist and distal phalanges remains. The sensory loss is variable; it may be absolute or absent. If there is anæsthesia it is greater on the palmar surface, and will often be found also on the dorsal aspect of the extremities of the first two fingers.

The *Ulnar nerve* comes, through the inner cord of the plexus, from the last cervical and first dorsal roots, and its origin from the lowest part of the cervical enlargement gives it an important relation, since it is the first of all the brachial nerves to be affected by disease that ascends from the dorsal to the cervical region of the spinal cord. The nerve supplies the ulnar flexor of the wrist, the ulnar half of the deep flexor of the fingers, the muscles of the little finger, the interossei, some of the lumbricales, the adductor and inner head of the short flexor of the thumb. Its sensory area is the ulnar side of the hand, back and front, generally more extensive on the back (two fingers and a half) than on the front (one finger and a half). The course of the nerve, superficial behind the elbow and at the wrist, renders it liable to separate injury. It is often implicated in wounds of the forearm, and about the elbow-joint, by dislocations of the shoulder and elbow, enlargement of the elbow bones, and by fractures of the bones of the forearm. It is occasionally the seat of neuritis, and, like the musculo-spiral nerve, it is sometimes paralysed by pressure. At the elbow, the projecting bones preserve the nerve from direct pressure; although Duchenne describes paralysis in men who, while at work, rest the bent elbow on a hard substance, this mechanism is certainly rare. It is much more common for the nerve to suffer in long-continued flexion of the elbow, without external pressure; and from this cause the nerve is sometimes paralysed during sleep. I have three times seen a sleep palsy of the ulnar nerve. The tension may set up neuritis, and is especially effective if the resistance of the tissues to morbid influences is lessened by general ill-health. Many persons must have noticed that if, when out of health, they sleep with the elbow bent, they wake with tingling, and even loss of feeling, in the region supplied by the ulnar nerve, although this may not happen when they are in good health. If the ill-health is profound, as in an acute illness, local neuritis of considerable intensity may be set up. Thus a lady, a few days after childbirth, who was prostrated by a long, exhausting labour, noticed tingling in the side of the hand, and, whenever she bent her elbow, a sensation "as if she had knocked her funny-bone." Paralysis of the muscles supplied by the ulnar nerve followed in a few hours, and in a fortnight there was distinct wasting. When I saw her, six months later, the ulnar nerve behind the elbow was distinctly thickened. She had had a similar, but more transient attack after a previous confinement. A similar palsy has occurred during the course of typhoid fever, and both nerves have been known to suffer (Bernhardt). In such conditions, not only is the tissue-health lowered, but warning sensations are often unperceived.

The importance of the interosseal flexion of the fingers for many actions, such as writing, renders palsy of the ulnar nerve peculiarly disabling. Several illustrations of the symptoms have already been given (p. 36). In flexion of the wrist, the hand deviates towards the radial side, and persistent distortion may take place. Adduction of the thumb is lost, and so are most movements of the little finger. The fingers cannot be flexed at the first or extended at the other joints, but the loss is slighter in the first two fingers than in the others, because their lumbricales are supplied by the median nerve. In time the opponents of the interossei, by their contracture, lead to over-extension of the first phalanges and flexion of the others; the "claw-like hand" is produced (see p. 36), but this is less complete than in cases of progressive muscular atrophy, on account of the escape of the first two lumbricales. When the palsy is imperfect it may be possible to extend the second and third phalanges, if the first can be over-extended, and an advantage thus given to the interossei by the lengthening of the course of their tendons, just as in partial paralysis of the long extensors they may be able to extend the digits if the wrist is flexed, but not if it is extended. In lasting damage the hypothenar eminence may disappear, the palm is hollow, the thenar muscles that remain stand out, and the opponens may cause a slight forward rotation of the thumb.

The loss of sensation, as in the case of the other nerves, varies much, both according to, and irrespective of, the degree of lesion of the nerve. Subjective sensations are common, with and without anæsthesia, and those who have had neuritis are sometimes unable to bend the elbow for a few minutes without a sensation of tingling in the area supplied.

The diagnosis of disease of the ulnar nerve is easy. A difficulty is occasionally caused by the circumstance already mentioned, that disease at the lowest part of the cervical enlargement may cause symptoms limited to the region of this nerve, but a knowledge of the fact is usually sufficient to prevent an error.

COMBINED PALSIES OF THE NERVES OF THE ARM.—Paralysis in the region of two or more of the nerves of the arm is very common, and results from many causes. It may be due to disease of the spinal cord or the nerve-roots within the spinal canal, but with these we are not now concerned. The most frequent causes outside the spinal canal are the following:—(1) Morbid processes in the neck, affecting the nerve-roots outside the spine or the upper part of the brachial plexus, especially growths and exostoses. (2) Diseases of the plexus itself, neuritis or injury, dislocations of the shoulder, the stretching by a sudden wrench of the arm,* or actual rupture of the nerves entering the plexus by great local violence. (3) Fractures of the bones. (4) Ascending neuritis, "neuritis migrans," which, commencing in a single nerve, may ascend to the plexus, and there spread.

We may consider first the cause last named. An injury of a single nerve is sometimes followed by an extension of the motor and sensory symptoms to the regions supplied by other nerves. This can only be explained by an ascending neuritis reaching the junction of the nerves at the brachial plexus, and there spreading, often in what seems a random manner. Thus a lady cut her wrist so as to leave a scar an inch long, over the position, and in the direction, of the median nerve. The injury caused paralysis and wasting of all the muscles in the hand supplied by this nerve. This palsy was followed by a gradual loss of power, with diminished faradaic irritability, in the long extensor of the fingers, the ulnar flexor of the wrist, and also by defect of sensation in the skin supplied by the ulnar nerve. Hence the neuritis must have ascended to the plexus, and there have spread along each root of the median nerve. Again, a woman cut her right hand with a broken stone bottle, along the hypothenar eminence. Three weeks afterwards the muscles of the hand supplied by the ulnar nerve gradually became paralysed, with wasting and loss of irritability, and in the area of the skin supplied by the nerve there was first tingling and then diminished sensibility. Burning pain spread up the front of the forearm, and inner side of the upper arm, to the axilla; all the forearm muscles lost power, the flexor carpi ulnaris, and the flexor sublimis digitorum in greatest degree; the tingling and lessened sensibility spread to the fingers supplied by the median nerve, and the abductor pollicis wasted. In this case also there must have been an ascending neuritis of the ulnar, spreading to the median at the brachial plexus.

The brachial plexus is occasionally the seat of a primary neuritis. This is described in a separate section (p. 97), as it is a defined and important disease. Another cause of damage to the plexus is dislocation of the shoulder, especially sub-coracoid dislocation, in which the displaced head of the humerus necessarily compresses the nerves. The extent of injury varies greatly; only one nerve may suffer, or not one may escape. Every muscle of the arm may be paralysed, from the deltoid downwards. In most cases the injury is severe in degree; there is rapid wasting of the muscles, with the reaction of degeneration, and there is a great tendency to the occurrence of trophic changes in the skin. It was in a case of this character that the forearm and hand were covered with blisters from the application of water that seemed merely warm to a healthy hand (p. 62).

Fracture of the humerus may damage both the musculo-spiral and ulnar nerves, rarely the median. Fracture of the bones of the forearm often injures both the median and ulnar nerves.

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* Bowlby, loc. cit., p. [illegible]

Injuries to the neck sometimes cause a partial paralysis of the arm of peculiar distribution, the special characters and significance of which were first made known by Erb.* A similar paralysis may come on apart from injury. The muscles affected are the deltoid, often the supra-spinatus and infra-spinatus, the biceps and brachialis anticus, and the supinators. Erb found that there is one spot between the scaleni, corresponding to the sixth cervical nerve, at which electrical stimulation puts all these muscles in action. Hoedemaker, who has described two cases of this palsy, finds the motor point in a line drawn from the sterno-clavicular articulation to the seventh cervical spine, 1·5 centimetres from the edge of the trapezius. The palsy is apparently dependent on disease of the roots of the fifth and sixth cervical nerves, and the fifth, it will be remembered, receives a twig from the fourth.† Besides injuries, this group of palsies may result from

FIG. 55.

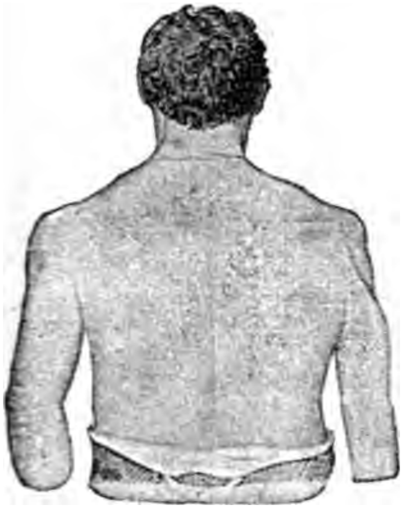
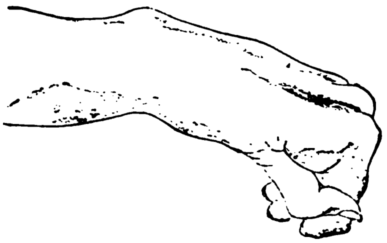


FIG. 56.



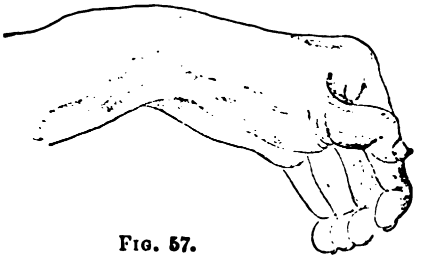


FIG. 57.

FIG. 55.—Combined palsy of deltoid, supra-spinatus, and infra-spinatus, from a fall on the shoulder.

FIGS. 56, 57.—Left hand of a patient suffering from a growth beside the lower cervical spine compressing the nerve-roots. There was anæsthesia of all parts supplied by the brachial and cervical plexus. The arm was adducted, the elbow flexed, the hand in the posture shown, flexion of first, extension of second, strong flexion of last phalanges, the first phalanx of the thumb over-extended, the second flexed. Rigidity extreme, and an attempt to overcome it caused great pain. There was also some contracture of the foot (equino-valgus).

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* Heidelberg Society, 1874, 'Ziemssen's Handbuch,' 1874, Bd. xii, pt. 2, p. 509; see also Bernhardt, 'Zeitsch. f. kl. Med.,' 1882, Bd. iv, p. 415. A previous description by Duchenne had escaped notice.

† Erb referred the palsy to disease of the sixth nerve. The experiments of Ferrier and Yeo point to the fifth and fourth roots as those innervating these muscles but his subsequent correction indicates the fifth and sixth (see p. 80).

non-traumatic processes, probably from neuritis, and also from growths in this situation. Duchenne and Seligmüller have also described paralysis of similar distribution produced in infants during birth, either by pressure from the position of the arm, or by traction on the neck with the finger or hook. Most obstetrical cases slowly recover. Those due to injury in adult life are often severe; the symptoms continue for a long time, and may be permanent.

Morbid processes connected with the bones of the cervical spine sometimes produce combined symptoms of irritation and palsy of the nerves of the arm, pain, hyperæsthesia, anæsthesia, paralysis of muscles, and extreme muscular contractures, often very irregular in distribution (such as are shown in Figs 56, 57). I have several times known sensory symptoms in the arm to be due to a chronic syphilitic cellulitis beside the vertebræ, causing deep-seated induration and severe compression of the nerves; in each case it was completely removed by anti-syphilitic treatment. Growths at the part may also compress the subclavian artery and the weaker pulse may aid the diagnosis.*

Diagnosis.—The diagnosis of diseases of the nerves of the arm has been for the most part sufficiently considered in connection with the special nerves. It consists chiefly in an application of the facts there stated. One or two more general considerations deserve, however, a brief mention. Some diseases of the spinal cord are first manifested in the arm, by muscular palsy, wasting, or by anæsthesia. The risk of error is prevented, in most cases, by the absence of any correspondence between these symptoms and the functions of special nerves, by the absence of any indication of morbid processes in the neighbourhood of the nerves, and by the presence of other signs of disease of the spinal cord. It has been mentioned that disease in the lowest part of the cervical enlargement may be manifested only in the region of the ulnar nerve, the lowest in origin of all the brachial nerves. Such disease is, however, usually bilateral, and secondary to disease in the dorsal region of the cord, the indications of which have preceded the symptoms in the arm. A knowledge of these facts, and a careful consideration of the distribution and course of the symptoms, will rarely leave the observer in doubt.

Treatment.—There is little in the treatment of the diseases of the nerves of the arm that requires special mention. The chief measure is the treatment of the cause of the paralysis. Any present source of pressure must be removed as far as possible. The ends of a divided nerve must be sutured. It is remarkable how quickly this has sometimes been followed by the restoration of conducting power. The posture of the arm must, in all cases, be such as to avoid tension on an injured or inflamed nerve. The treatment of neuritis is described in the next section. Electrical stimulation of the muscles is of great importance in all cases of severe damage to the nerves. Even in old and stationary cases it sometimes starts some improvement.

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* Bowlby, loc. cit.

NERVES OF THE LOWER LIMB.

Disease of these nerves is far less common in the lower than in the upper limb, with one important exception,—the primary disease of the sciatic nerve that goes by the name of "sciatica." This affection is reserved for separate description in the next section.

The nerves of the leg are derived from the lumbar and sacral plexuses. The *lumbar plexus*, consisting of the first three and a half lumbar roots, supplies the skin of the lower part of the abdomen, of the front and sides of the thigh, of the inner side of the lower leg and foot. It supplies also many muscles—the cremaster, and those that flex and adduct the hip-joint, and those that extend the knee. Its branches for the leg are the obturator and anterior crural nerves. The *sacral plexus* consists of the fifth lumbar root and half the fourth (lumbo-sacral cord) and the first four sacral nerves, of which, however, only the upper three have to do with the leg. This plexus innervates the extensors and rotators of the hip, the flexors of the knee, and all the muscles that move the foot, together with the skin of the gluteal region, the back of the thigh, the outer side and back of the lower leg, and most of the foot. Its chief nerves are branches to the outward rotators of the hip, the gluteal nerve, and the small and great sciatic.

The results obtained by Ferrier and Yeo (see p. 80) on stimulating the roots of the lumbar and sacral plexuses in the monkey, may be thus summarised:

Lumbar I and II. Lower abdominal muscles (not cremaster).
III. Psoas and iliacus, sartorius, extensors of knee; (flexion of hip and extension of leg).
IV. Glutei, adductors, extensor cruris, peroneus longus; (extension of hip and knee, elevation of outer side of foot).
V. Glutei, hamstrings, and all the muscles in front and back of lower leg; (rotation of thigh outwards, flexion and rotation inwards of leg; extension of foot with elevation of outer edge; flexion of distal phalanges of toes).
Sacral I. Hamstrings, calf muscles, long flexor of toes, intrinsic muscles of foot; (slight outward rotation of thigh, flexion of knee, extension of foot, adduction of great toe, flexion of first phalanges of all toes and of both phalanges of great toe).
II. Intrinsic muscles of foot; ("interosseal" flexion of toes, similar to the last).

These results cannot be simply applied to man. For instance, it is certain that in man, one flexor of the hip, the psoas, and the cremaster, are largely innervated from the second lumbar, but no indication of the action of either of these could be observed on stimulating this root in the monkey.

The distribution of the sensory fibres of the nerve-roots is considered in the chapter on the Spinal Cord.

The *Lumbar plexus* itself is sometimes damaged by growths in the abdomen (especially by those that spring from the lumbar glands) and by psoas abscess, while the nerve-roots may suffer in disease of the bones of the vertebræ and in meningitis. The plexus may also be invaded by inflammation ascending the lumbo-sacral cord from the sacral plexus, and it is occasionally the seat of spontaneous neuritis. The *obturator nerve* is rarely affected alone; it has been sometimes damaged in the course of parturition. The *anterior crural nerve* has suffered from the same cause, and is sometimes injured by wounds of the groin or thigh, or by dislocation of the hip-joint. Symptoms may be limited to it in disease of the nerve-roots.

Of the interference with movement that may result, the paralysis of the flexors of the hip depends on the position of the disease. This paralysis is total only when the lumbar plexus is damaged. Disease of the *anterior crural* nerve, within the abdomen, does not affect the psoas, but may paralyse the iliacus, and so weaken, without abolishing, the power of flexion. The chief symptom of disease of this nerve is the loss of power in the extensors of the knee, and the wasting of these muscles, together with loss of the knee-jerk which results from the interruption of the reflex arc. The effect of these palsies on movement is very serious (see pp. 40, 41). Anæsthesia involves the whole of the thigh (except a strip along the middle of the back), the inner side of the leg and foot, and the adjacent sides of the first and second toes. In many cases the paralysis is incomplete, and the symptoms present corresponding variation. Irritation of the nerve may cause severe pain in the region supplied by it. This is sometimes an early symptom of a growth near the spine. The pain in neuritis of the plexus may extend along the course of the ilio-hypogastric, ilio-inguinal, and genito-crural nerves, to the lower part of the abdomen and groin.

Paralysis of the *obturator* nerve causes a loss of the power of adduction of the thigh, so that the patient cannot put one leg across the other. Rotation of the thigh outwards is interfered with, but in slighter degree than adduction. The effect of these palsies (described on pp. 39, 40) is far less serious than is the disability which results from disease of the anterior crural.

The *superior gluteal nerve* occupies an intermediate position between the two plexuses, arising as it does, from the lumbo-sacral cord, which descends from the fourth and fifth lumbar roots. Its disease, which is very rare in isolated form causes paralysis of the gluteus medius and minimus, with a loss of abduction and circumduction of the thigh (see p. 39).

The *Sacral plexus* is sometimes damaged by growths in the pelvis, by pelvic inflammation of various kinds, and by compression during parturition. It is also often the seat of neuritis, which, however, less frequently begins in the plexus, than ascends to it from the sciatic nerve. Apart from spontaneous neuritis, which will be separately described, the sciatic nerve outside the pelvis is occasionally injured by wounds, rarely by dislocations of the hip, often by disease of the bone, and by adjacent morbid growths. It is also a relatively frequent seat of neuroma. Of the terminal branches, the external popliteal, by its superficial course, and proximity to the hard bone, suffers from traumatic lesions of various kinds; it is also prone to spontaneous neuritis. This nerve is homologous with the musculo-spiral nerve of the arm, and presents an analogous liability to disease. The posterior tibial nerve is more secure in its deeper course, but may be damaged by fracture of the bones.

The symptoms of palsy of the *sciatic* nerve vary much in their character, according to the position of the disease. The *small sciatic* is implicated only when the mischief involves the pelvic plexus, and it scarcely ever suffers alone. The effect is palsy of the gluteus maximus, which interferes with rising from a seat more than with walking (see p. 39). There is also anæsthesia of the skin in the middle third of the back of the thigh, and in the upper half of the calf. A lesion of the sciatic nerve, near the sciatic notch, paralyses the flexors of the leg (which are also extensors of the hip; see p. 42), and all the muscles below the knees. Often the lesion is below the upper third of the thigh, and then the flexors of the leg escape. Even when all the muscles below the knee are paralysed, walking is still possible, the foot being raised by over-flexion of the hip. The anæsthesia that results from a lesion of the nerve below the origin of the small sciatic, involves the outer half of the leg, the greater part of the dorsum of the foot, and all the sole, but the leg may escape, perhaps through union with other nerves.

The symptoms of disease of the branches are as follows:—

That of the *external popliteal* or *peroneal nerve* causes paralysis of the tibialis anticus, long extensor of the toes, peronei, and extensor brevis digitorum. The effect of this is a loss of all power of flexing the ankle and of extending the first phalanx of the toes (see p. 44). The foot cannot be raised from the ground in walking, and talipes equinus ultimately results (Fig. 30, p. 44), which may be attended with persistent flexion of the first phalanges of the toes from contracture of the unopposed interrossei. There is also anæsthesia on the outer half of the front of the leg, and on the dorsum of the foot.

Disease of the *internal popliteal nerve* paralyses the popliteus, calf muscles, tibialis posticus, and long flexors of the toes, as well as the muscles of the sole. In addition to the disability which characterises paralysis of the plantar muscle, there is loss of the inward rotation of the flexed leg, if the disease is so high as to involve the branch to the popliteus, and there is also loss of the power of extending the ankle-joint. Talipes calcaneus results (Fig. 28, p. 43). The sensory loss is on the outer lower part of the back of the leg, and on the sole, but varies much.

The *plantar nerves* rarely suffer alone. A lesion of the *internal nerve* causes anæsthesia on the inner part of the sole, and plantar surface of the three inner toes and half the fourth, together with paralysis of the short flexor of the toes, the plantar muscles of the great toe (except the adductor), and of the two inner lumbricales.

Disease of the *external nerve* produces anæsthesia of the skin on the outer half of the sole, the little toe and half the fourth, paralysis of the flexor accessorius, the muscles of the little toe, all the interossei, the two outer lumbricales, and the adductor of the great toe. The effect of this palsy (see p. 45) is serious, since the toes cannot take their proper share in propelling the body forward in walking, and they gradually become flexed at the last two joints and extended at the others, from the contracture of the opponents of the interossei,—a position of the toes that causes serious inconvenience in walking.

DIAGNOSIS.—The diagnosis of diseases of the nerves of the leg is determined by the same general principles as those that have been mentioned as applicable to the nerves in general, and to the nerves of the arm in particular. The limitation of the symptoms to the functional areas of individual nerve-trunks, the evidence afforded by nutrition, irritability, and reflex action that the muscles are separated from the spinal cord, the implication of the sensory functions, and often the tenderness of the affected nerves, indicate, in most cases, the seat of the disease. To these signs, are often added other indications of a local cause, corresponding, in its position, with the nerve to which the symptoms point.

The relation of nerve-trunks to nerve-roots, although by no means simple, is certainly less complex in the case of the nerves of the leg than in those of the arm. This is especially the case in the lumbar plexus, and it leads to an occasional difficulty in diagnosis. Pressure on the spinal cord, for instance, at the level of the origin of the fourth lumbar roots, may cause symptoms identical with those of a partial lesion of the anterior crural nerve. For instance, I have known paralysis of the extensors of the knee, loss of the knee-jerk, with anæsthesia in the front of the thigh, to be the result of a gumma at the side of the cord at this level. But other evidence of a spinal lesion is rarely absent under such circumstances, and, in this patient a foot-clonus, due to the pressure on the pyramidal fibres, left no doubt as to the situation of the disease. Another difficulty arises from the long course of the nerve-roots in the cauda equina, disease of which may simulate that of the nerves of the leg. But the symptoms are commonly bilateral in consequence of the proximity of the nerve-roots of the two sides. In all cases in which symptoms are bilateral (unless there is evidence of a disease known to cause symmetrical lesions, such as multiple neuritis), the suggestion is that the disease is situated where the motor or sensory paths of each side are so near that they can be affected by a single lesion, i. e. that the disease is within the spinal canal. But here, as in other cases, we cannot reverse our diagnostic rules. Disease of the spinal cord does not always cause bilateral symptoms. A limited lesion of one anterior cornu may be so placed as to paralyse the muscles supplied by a single nerve, and a doubt may be felt as to the central or peripheral origin of such palsy. The muscles supplied by the anterior crural nerve, and the muscles in the front of the lower leg supplied by the external popliteal, are those of which the central palsy most often leads to doubt. The mode of onset, the presence or absence of sensory symptoms, the rarity of acute spinal palsy except in childhood, and of nerve-lesions except in adult life, the wider initial prevalence of the palsy in acute, and its later extension in chronic, cornual disease,—these suffice as a rule to remove any doubt.

It is important to remember that the pressure of a growth may cause either a chronic or an acute affection of the nerves. The chronic symptoms result from compression; the acute from a neuritis set up by the pressure and irritation.

Treatment.—The treatment of disease of the nerves of the leg does not differ from that of disease of the nerves of the arm. More care, perhaps, is needed to avoid increasing present mischief, or inviting a relapse, by exposure to cold or by fatiguing exertion. More care is also needed to obviate the tendency to secondary contractures in the case of palsies of long duration, and in those attended by pain, in which the patient seeks ease in postures to which the muscles only too readily adapt themselves. The contraction of the hamstrings, from constant flexion of the knees, occurs very readily and is most troublesome; that of the calf muscles, which occurs when the flexors of the ankle are paralysed, also constitutes a serious obstacle to walking after recovery. A little timely care, by attention to posture, will often save a vast amount of later trouble. That of the calf muscles, however, which is due to the extension produced by the weight of the foot, as the patient lies, cannot always be entirely prevented, but may be lessened by a board or large sand-bag against which the feet can rest.

BRACHIAL NEURITIS.

Besides the forms of inflammation of the nerves of the arm already described, a primary inflammation of the brachial plexus occurs in a form as well defined as sciatica, and equally meriting a separate description. This, however, need only include its special features; those that are common to other forms of neuritis have been already described. It is so closely analogous to sciatica that it may be called "sciatica of the arm." It is essentially a perineuritis,—a primary inflammation of the sheaths of the branches that enter and form the brachial plexus.

Cases occur, however, in which the symptoms correspond in general character to the rest, but in which their distribution suggests that the nerve-roots rather than the plexus are the seat of the inflammation, and pain about the spine supports the opinion. This form may be called *Radicular Neuritis.* Although its existence has not been established by pathological evidence, the symptoms mentioned admit of no other explanation. A knowledge of their significance is of great practical importance on account of the closeness with which they may simulate those of organic disease of the bones of the spine, or a growth in the spinal membranes.

Causes.—The influence of gout in causing local neuritis is very conspicuous in the brachial form, with the special characteristic that this occurs chiefly late in life, very often from the inherited disease, and with greater frequency in females than any other form of neuritis; their liability is at least equal to, if not greater than, that of men. Five-sixths of the cases occur after fifty, and it may be met with up to extreme old age. In men there have usually been the ordinary manifestations of gout, but in women the tendency is often only indicated by the family history, and by previous muscular rheumatism, especially lumbago and sciatica,—which have seldom been absent in either sex. In one case the onset of the brachial neuritis occurred immediately after an attack of sciatica.

Symptoms.—Pain, the great symptom of the inflammation of all mixed and sensory nerves, is greater in this than in most forms of neuritis. It is usually the first symptom, and lasts long after the inflammation is over; its severity, coupled with the age of most sufferers, renders the malady one of a peculiarly trying character. The first pain is often referred to a distance from the seat of the inflammation, perhaps because this begins at the plexus, where, at divisions, it is facilitated by motion, and the conducting fibres are readily reached and early irritated. Frequent seats for the first pain are the region of the scapula (sometimes beneath the bone) and the wrist or back of the forearm, with or without the hand. In other cases, however, the first pain is at the plexus itself, above the clavicle or in the axilla, and these are the places in which it is commonly most intense throughout the attack. As the pain increases it extends along the course of the nerves of the arm, which the patient will often accurately indicate with his finger when tracing the lines of pain.

The pain is sometimes sudden in onset and severe from the beginning; more often it is at first occasional, or felt on certain movements, but, as it increases, it becomes more continuous, with variations that soon rise to paroxysmal degree. Ultimately there is always more or less dull wearying pain in the whole arm, but especially in the region of the plexus, varied by attacks of great severity. In these the pain is acute and lancinating, or stabbing, or burning; it usually takes the course of the nerves, diffusing itself from them, and often passing to the side of the chest, and to the neck,—seldom to the head. The pain often varies in character according to intensity; at the height of the paroxysm it may be sharp and stabbing, or such darting pains may be superadded to a more diffused burning pain, which lasts longer than the acute pain, and may be followed, as the paroxysm subsides, by general tingling of the skin of the whole limb. The paroxysms are induced by movement or occur spontaneously. In slighter cases the pain is paroxysmal only, and then the relation to movement is a very conspicuous feature. Although it is seldom confined to movement, this never fails to induce severe pain, and the patient avoids the slightest use of the limb. Elevation of the arm especially causes distress.

With the pain there is usually undue sensitiveness of the skin, which may be much increased during and after the paroxysms. Loss of sensation is rare, and is met with only in cases that are not only severe but prolonged. The two may concur, as "anæsthesia dolorosa."

The muscles usually present the flabbiness and slight wasting common in neuritis, but the damage to the motor fibres is seldom sufficient to cause considerable atrophy. Sometimes, however, there is enough damage to cause wasting of some group of muscles, with the reaction of degeneration. This is most frequent in the radicular form, in which the damage is to the upper part of the plexus and nerve-roots. There may then be anæsthesia of the skin over the affected muscles. It is difficult to ascertain the existence or the amount of motor weakness; effort induces pain so readily that the patient can seldom be induced to make an attempt to exert force. Power is often said to be almost lost, when it is probably greater than is believed. Besides the muscles, the subcutaneous tissue of the limb may also waste, and the skin may become thin and shining, and present the aspect already described. Subcutaneous œdema is also common. Arthritic changes in the joints of the fingers are almost constant in the cases that occur in later life; the adhesions may be permanent.

Diagnosis.—Few maladies, as a fact of experience, give rise to greater diagnostic difficulty. This is due to several causes: the affection is rare; its symptoms are sometimes equivocal; the subjects are usually in the degenerative period of life, when many obscure diseases attended with pain in the arm occur to the mind of the physician; and lastly, the distinction between neuralgia and neuritis is often difficult, although less so than is supposed. The last is indeed the most frequent source of error. The most severe and characteristic cases of brachial neuritis are frequently mistaken for pure neuralgia, on account of the paroxysmal character of the more severe pain, and because the characters of neuritis are unfamiliar. The points to determine the diagnosis are those described in the sections on neuritis and neuralgia;—the degree of persistent tenderness of the nerves and the influence of movement, together with the history of the attack, the locality of the pains, and especially any evidence of damage to the fibres. A history of neuralgia in the person or the family is of slight weight only; the tendencies to true neuralgia and to neuritis are often combined, and each is a frequent result of gout. A far greater difficulty is presented by the cases in which the inflammation remains slight and is confined to the plexus, affecting branches where the conducting fibres can be so irritated as to cause distant pain, which then becomes the leading symptom. This is often paroxysmal, and, when on the left side, may radiate to the chest and be associated with disturbed action of the heart. Angina pectoris is often thought of in such cases; and here again the degenerative age may increase the difficulty by leading to the presence of some coincident disease of the heart, or by making true angina not unlikely. The distinction afforded by the nerve-tenderness is then of great importance, because the disproportionate amount of tenderness (compared with the amount of pain) is more emphatic in slight than in severe cases. Persistent tenderness with only paroxysmal pain should always suggest neuritis. When the position of the pain is carefully examined, its relation to the plexus and branches is often clear. It is important to remember that all nerve-pains in the brachial region on the left side have a tendency to resemble anginal pain in distribution, and to be associated with cardiac distress. Probably there is some peculiar tendency for pains in this part to disturb the action of the heart; a common physiological relation may underlie both the nerve-pains of cardiac angina and the cardiac symptoms of nerve-pains. Hence this secondary disturbance does not neutralise the significance of the special signs of neuritis. In some cases of the slighter class, the pains suggest the idea of an aneurism; in many cases of brachial neuritis this diagnosis has been made, and the patient has had to endure months of mental distress, for which no real cause was in existence. Such a suspicion, in the absence of special signs, such as pressure-symptoms, should only be entertained if the pains are persistently and increasingly severe, and unaccompanied by any considerable amount of tenderness. In all these cases, moreover, the presence of the gouty diathesis may be allowed weight in the diagnosis.

The muscular wasting, slight in degree, added to the arthritic changes, gives rise to a condition which may be mistaken for a primary joint affection with secondary "arthritic atrophy" of the muscles. It is only in the chronic stage that this error is possible. A careful attention to the history of the case will show its real nature, but without this attention the mistake is easy, and it is often made.

PROGNOSIS.—Except in its most trifling degree, brachial neuritis is a tedious malady; the duration of every severe case is to be measured by months, and often more than a year elapses before the patient is free from pain. Post-neuritic pain is always more prolonged in the old than in the young, and the age of the subjects, together with the amount of damage to the nerves, causes suffering to last longer than in almost any other form. To these causes also must be added the degree of sensitiveness of the affected nerves, exceeded only by that of the fifth nerve, and also the mobility of the parts in which the plexus lies; this involves a continued cause of irritation, brought into play as soon as the diminution in the severer pain permits the patient to employ the long useless arm. Relapses, moreover, are not uncommon. Recurrence may take place after moderate attacks, but seldom occurs after those of extreme severity.

Recovery from the consequences of the inflammation is not always complete. The limb often remains smaller and feebler, with a liability to tingling and to cramp, and also to neuralgic pains under the influence of changes in the weather, &c. But the most serious consequences are the alterations in the joints. These are very frequent: the fixation occasioned by the pain permits, and the tendency to perverted nutrition produces, adhesion between the articular surfaces, in consequence of which the movement becomes permanently restrained. It is probable, moreover, that the constitutional state which underlies the primary malady often increases the degree of these joint-alterations. The shoulder, wrist, and fingers are the joints that are most frequently thus rendered stiff. The interference with the movement of the fingers is especially troublesome; the joints become painful in consequence of the unavoidable strain on the tissues when an attempt at use is made.

Treatment.—The treatment that has been described, as needed in neuritis generally, is suitable also for this form, and its details need not here be repeated. Abstinence from movement is of great importance; the occurrence of stiffness of the joints in consequence of the immobility of the limb must be risked. It is doubtful, indeed, whether the tendency to this is much increased thereby, for the effect of pain, in preventing sufficient movement to maintain the normal state of the joints, is not materially increased by rest otherwise enforced; while the difference between a little movement and none, during the acute stage of the affection, is enough to make a considerable difference in the effect on the inflammation of the nerves. The influence of cocaine, injected under the skin, is very beneficial, but it is needed frequently in severe cases, and two injections (each $\frac{1}{10}$—$\frac{1}{8}$ gr.) may be given daily during the height of the disease. It is important that the nerves should not be irritated by massage in the state of active inflammation; indeed, no therapeutic measure which occasions pain can do as much good as harm. After the tenderness has subsided, gentle rubbing, such as is agreeable, may be employed with advantage. Electricity is only needed for muscles that present the reaction of degeneration, or to aid in removing after-pains.

SCIATICA.

As the word sciatica is commonly used, it is a general designation for all affections of which the chief symptom is pain in the region of the sciatic nerve. It is thus often applied to the pain which results from pressure on the nerve in the pelvis, as by a tumour. In a stricter use of the word, however, it is applied to painful affections of the nerve not due to any cause outside it. The two varieties may be distinguished as secondary and primary sciatica. Primary sciatica is commonly regarded as a neuralgia. This view is in the main erroneous; the vast majority of cases of sciatica are really cases of neuritis of the sciatic nerve. The reasons for this opinion are given in the section on the pathology of the disease.

Causes.—Sciatica is far more frequent in males than in females; the proportion has been very variously estimated. It is difficult to obtain statistics on the malady that are free from sources of fallacy, since comparatively few cases enter general hospitals. The incidence of severe sciatica seems to be fairly presented by the 137 cases that were treated in the Devonshire Hospital, Buxton, during the year 1883, and there is no reason to suppose that slight cases differ, in this respect, from those that are severe. The percentage of males is 82; of females, 18; giving a ratio of 4 to 1, which is probably very near the truth—a remarkable contrast to the analogous affection of the arm. The disease is unknown in childhood, and rare in the second decade of life. It is most frequent between forty and fifty, next between fifty and sixty, and next between thirty and forty. The percentage distribution of the 137 cases at Buxton is as follows:—Ten to twenty, 3 per cent.; twenty to thirty, 9 per cent.; thirty to forty, 19 per cent.; forty to fifty, 29 per cent.; fifty to sixty, 26 per cent.; sixty to seventy, 13 per cent.; over seventy, 1 per cent. The influence of age is nearly the same in each sex.

Two constitutional conditions are potent factors in the production of sciatica—gout, and that form of rheumatism which affects chiefly the fibrous tissues of the muscles. These two constitutional states cannot be entirely separated, but many of the sufferers are of opposite types: some are stout, plethoric, distinctly gouty; others pale, weakly, and rheumatic. Many of the latter owe their rheumatism to inherited gout; rheumatic fever is certainly one of the effects of this family tendency. Sciatica is most common among those who are gouty but do not suffer from articular gout, and such individuals often have many attacks. Gouty inheritance is generally to be traced in those who suffer early from the disease. Syphilis has been supposed in some cases to be the cause of a sciatic neuritis, but the cases are so rare as to make strong and clear evidence desirable.

An exciting cause is to be traced in many of the cases. Exposure to cold is the most common. It is usually local exposure, as by wet boots, standing in water, &c.; sometimes, however, a general chill of the body determines an attack. The exposure to cold may be even more direct, as by sitting on wet grass. I believe that draughty water-closet seats are answerable for some cases. The neuritis often arises by the extension of an adjacent rheumatic affection of the fibrous tissue, especially of that form of lumbago which involves the fibrous attachments of the muscles at the back of the sacrum. This passes down, extending along the fasciæ, to the nerve-sheath in the neighbourhood of the sciatic notch. (The fact is of much interest, because it shows that this form of rheumatism must be regarded as inflammatory since it can spread as inflammation.) A few days after the pain reaches the nerve-sheath there may be all the signs of intense inflammation. Mechanical causes also sometimes excite the disease, and still more often co-operate with other influences. The pressure of the edge of the chair, in those who sit much, is the most frequent mechanical cause. Muscular over-exertion has been thought occasionally to excite the disease. If the nerve is already tender, a strong contraction of the muscles at the back of the thigh, especially when the knee is flexed, and the muscles can freely shorten and widen, may produce acute pain in the nerve, evidently by its compression. It is conceivable, therefore, that this cause may be effective in a predisposed person. Various morbid processes within the pelvis may cause symptoms of sciatica, usually by acting on the sacral plexus. Thus rectal and other tumours, pelvic inflammation, and injury during labour are occasional causes. A loaded rectum has been thought to be capable of producing sciatica. Mechanical congestion of the plexus of veins which lies over the sacral plexus of nerves is usually regarded as an occasional cause, but this is somewhat hypothetical, and its efficiency can scarcely be demonstrated. Lastly, the sciatic nerve may be secondarily involved in mischief that is outside the pelvis. The most frequent cause of this is disease of the bone, and especially disease of the hip-joint. In all these forms of secondary sciatica the symptoms are due partly to pressure on the nerve, partly to inflammation excited in it by the pressure, or extending to it from the adjacent disease.

PATHOLOGICAL ANATOMY.—Sciatica being a disease that does not itself cause death, observations of the morbid changes are few. In most cases that have been examined, distinct evidence of neuritis has been found, chiefly involving the nerve-sheath, but extending in some cases to the interstitial tissue. The changes are those that have been already described in the account of neuritis (p. 54). In recent cases there is swelling and redness of the sheath, sometimes there are small hæmorrhages, and, in severe cases, similar but slighter alterations in the instertitial tissue, with secondary damage to the nerve-fibres. The signs of inflammation are most intense at the sciatic notch, and opposite the middle of the thigh. They may be limited to one or both of these places, or they may be greatest there, and extend, in slighter degree, over a considerable tract of the nerve.

SYMPTOMS.—The chief symptom of sciatica is pain along the course of the nerve-trunk, often also along that of its branches, and sometimes in the area of its distribution. The affection may begin suddenly, especially in cases of rheumatic origin,—as suddenly as lumbago—generally about the hip or sacrum. Some sudden movement seems to excite it, but there has usually been slighter rheumatic pain in the neighbourhood for a day or two. More frequently the onset is gradual; slight pain is felt along the back of the thigh, on movements and in postures that make the nerve tense, or cause pressure upon it. As the disease increases, the pain is more readily produced, and becomes greater in degree, until at last the patient is free from induced pain only when at rest and the leg is in a certain position. Any movement that makes the nerve tense causes extreme suffering, and to avoid this the knee, in walking, is kept slightly flexed, and the leg held stiffly so as to avoid painful changes in the tension of the nerve. As the pain on movement increases, spontaneous pain is added, at first chiefly felt in the nerve-trunk, but soon spreading to its branches and distribution. It is usually most intense in certain spots—(1) above the hip-joint, near the posterior iliac spine, (2) at the sciatic notch, (3) about the middle of the thigh, (4) behind the knee, (5) below the head of the fibula, (6) behind the external malleolus, (7) on the back of the foot. The pain may radiate over the whole distribution of the nerve, but it is often so distinctly limited to the course of the trunk and branches, that the patient points these out with exactness when he indicates the course of the pain. It may be dull or acute, is often burning in character, and worse at night. It may seem to dart downwards, starting from the highest point. As the pain on movement increases, the nerve-trunk becomes extremely tender to pressure, especially along the back of the thigh. Even before the tenderness becomes considerable it may often be elicited in the following manner. Let the patient sit on a chair with the knee at a little more than a right angle. If the finger is then pressed into the popliteal space, so as to make the nerve a little more tense, a pain is felt in the course of the nerve at the back of the thigh or above the sciatic notch. It is due to the sensitiveness of the nerve to tension, and is a very useful test. It is of much diagnostic value, especially when the part inflamed is high up within the pelvis, and there is no tenderness to pressure at the back of the thigh.

Abnormal sensations other than pain are often felt in the area of distribution of the nerve,—tingling, formication, and the like; and in severe cases there may be irregular spots of anæsthesia on the back of the thigh, on the leg, or the foot. The affection of sensibility at the back of the thigh indicates that the disease extends up the nerve, above the sciatic notch, to the origin of the small sciatic, or that this is involved in a simultaneous neuritis. In severe cases the muscles supplied by the nerve become flabby, tender to the touch, and sometimes distinctly weak and wasted. This is chiefly noticeable in the calf muscles. There may be slight alteration in the electrical irritability, but this amounts to a distinct degenerative reaction only in very severe cases. Slight fever and corresponding constitutional symptoms may attend an onset that is acute, or an inflammation that is intense. Chronic cases, however, are usually not attended by elevation of temperature.

The duration and severity of the affection are extremely variable. It may be trifling in degree, causing pain on movement only, and this may pass away in the course of a few weeks. On the other hand, the spontaneous pain may be so continuous and intense that sleep can be obtained only by the help of narcotics, and the disease may last for many months, and even for a year. In most cases that last more than a year there is really a relapse of the affection. Improvement is shown by the subsidence of spontaneous pain, followed by the slow diminution of the pain on movement, and of the tenderness of the nerve. The muscular wasting, which occurs in severe cases, may last long after the active stage of the disease in the nerve; and fibrillary contractions in the muscles that have been affected may continue for years, and are sometimes accompanied by a tendency to painful cramp. For a long time after the attack is over, pain is felt in the nerve in certain postures. Occasionally a secondary neuralgia is set up, extending outside the sciatic area. The disease is prone to relapse, and still more prone to recur after recovery. A second attack may occur in the same or in the other leg, but both legs are scarcely ever affected at the same time. I have only once known a true primary sciatic neuritis to be bilateral.

The cases of secondary sciatica depending on disease outside the nerve, compressing or irritating it, differ, in some respects, from the primary form. Pain is felt less in the nerve-trunk than in its distribution. The difference is not absolute; it is one of degree. There is rarely, however, the tenderness of the nerve that is so common in primary sciatica. The course of the secondary cases is also different, and depends on the course of the original disease.

Complications of sciatica are rare, but one or two deserve mention. Cutaneous eruptions, usually herpetic in character, have been occasionally met with. These may heal slowly, but, in themselves, they are unimportant. Sometimes the epidermis exfoliates; œdema of the limb occurs in rare cases. A graver complication, but happily a rare one, is a tendency of the mischief to ascend the nerve. Thus symptoms may spread from the region of the sciatic to that of the lumbar plexus; the pain may spread to the front of the thigh, and the extensors of the knee may become flabby and weak. This is probably due to the passage of an ascending neuritis up the lumbo-sacral cord. In extremely rare cases the morbid process has apparently reached the spinal cord, and indications of cord disease have thus succeeded those of a primary and apparently simple sciatica.

PATHOLOGY.—The nature of the disease has been already indicated. The evidence that the disease is a neuritis, and not a neuralgia, is chiefly threefold. The conditions under which the disease occurs are very different from those that attend unquestionable neuralgia in other situations. This has been remarked with surprise, even by those who regard it as a neuralgia, but who have failed to see the significance of the difference. Secondly, the neuritic nature of the affection is clearly indicated by the facts of pathological anatomy, scanty though these are. Thirdly, the symptoms in severe cases are proof of the existence of neuritis, the wasting of the muscles and the anæsthesia indicate structural damage to the nerve-fibres, and preclude any other explanation. There is every gradation between these severe forms and those which are slight, and the symptoms in the latter are identical with those of the earlier stage of the severe cases.

The symptoms find their explanation in the character of the morbid process, and the facts of neuritis in general, already described, apply to this disease in every point. It is primarily a perineuritis; the pain in the nerve and its tenderness are due to irritation of the sheath-nerves. The pain referred to the distal portions of the nerve is due to the irritation of the proper fibres by the interstitial inflammation, while their greater damage explains the anæsthesia and muscular wasting.

DIAGNOSIS.—The diagnosis of sciatica rests on the position of the pain—on the relation of this to the trunk of the nerve, and to its area of distribution. But the recognition of this relation does not alone suffice for the diagnosis. We have to consider the distinction from other pains in the sciatic area, to discriminate, if we can, sciatic neuritis from sciatic neuralgia, and to decide whether the affection of the nerve is primary, or is secondary to mischief outside it. It should be needless to say that pain felt only on the outer side of the thigh is not sciatica, although the name is sometimes applied to such cases, apparently because, some name being needed, that of the nearest affection is chosen.

Branches of the sciatic nerve and sacral plexus ramify over the hip-bone, and pain in sciatica may be felt near the hip-joint; hence disease of this joint and sciatica may be confounded. The pain in simple disease of the hip-joint does not extend down the back of the thigh in the course of the sciatic, and there is no tenderness of the nerve. The danger of this error is not great if a careful examination is made, and has been much exaggerated by writers unfamiliar with the characteristic symptoms of true sciatica.

The distinction between a sciatic neuritis and a sciatic neuralgia is sometimes difficult, although less frequently than might be inferred from current accounts of these diseases, in which the history of the neuralgia has been written from the symptoms of the neuritis. If we recognise that all cases of sciatica with persistent tenderness of the nerve are really neuritic, cases of sciatic neuralgia become extremely rare. The two diseases occur usually under different conditions: the subjects of neuralgia have often suffered from neuralgia elsewhere, and are generally weakly and anæmic. The pain is from the first spontaneous; posture has little influence upon it; movement is not itself painful, although it may excite paroxysms of pain. The pain is referred to the branches and distribution of the nerve rather than to its trunk, or it darts up or down the trunk; and tenderness of the nerve, if it exists, is altogether subordinate to the spontaneous pain.

Secondary sciatica is usually produced by disease of bone about the hip-joint, or of the joint itself, or by disease in the pelvis. In the former case a careful examination (never to be omitted in any case of sciatica) at once reveals the mischief. When the disease is within the pelvis, the tenderness of the trunk of the nerve is slight in proportion to the pain, and this circumstance should always lead to a careful search for any indication of pelvic mischief. In any case of doubt a rectal examination should be made.

Sciatic pain occurs in some diseases of the bones of the spine, in lesions of the cauda equina, and occasionally in disease of the spinal cord itself. In these cases we have little or no tenderness of the nerve; the pain is chiefly peripheral, and very often bilateral. Double true sciatica is so rare that bilateral pain should always suggest disease of the nerve-roots. The pains of locomotor ataxy are often felt in the sciatic area, and occasionally follow the course of the sciatic nerve; but their wider range, their fugitive character, and their association with other symptoms of tabes, should prevent an error.

PROGNOSIS.—The prognosis of sciatica, not dependent on disease outside the nerve, is always good as regards ultimate recovery. As a general rule, the probable duration of the disease is proportioned to the severity of the symptoms. The practicability of adequate rest is an important element in the prognosis. Irritating exertion may lengthen the duration of the disease by many months, and indeed relapse may follow each partial recovery until one or even two years may pass before the sufferer at last becomes free from pain. When the nerve is so sensitive that the patient cannot stand, the affection will continue for some months.

TREATMENT.—The principles of the treatment of sciatica are those of neuritis already described, and only the points of special importance need be here repeated. In all cases rest to the limb is essential, and its urgency is proportioned to the acuteness and severity of the symptoms. Many slight cases are converted into severe ones by unwise exertion. All postures and all movements which increase pain should be avoided. The same principle applies to mechanical compression of the nerve by hard seats, and by strong contractions of the flexors of the knee. In more positive treatment the possible causes of the disease must be remembered. In gouty cases saline purgatives are often of signal service, and are distinctly useful in preventing attacks in those who are liable. In the acute stage of a severe attack, hot linseed-meal poultices should be applied along the course of the nerve. Counter irritation is of great value, and cannot be employed too early. A commencing attack may often be cut short in a few days by rest, and a series of mustard plasters or small blisters applied over the seats of pain, as this changes under their influence, chasing it, as it were, from one spot to another, until it disappears. Internally, whenever there is reason to believe that active inflammation exists, mercury should be given—a grain of blue pill twice daily; nothing else seems distinctly to influence the process. Salicylate of potash or lithia and nitrous ether should also be given at the onset—soda salts being avoided. Spontaneous pain can only be relieved by sedatives. Morphia is the surest, but it should only be used for the relief of severe spontaneous pain. In no malady has cocaine proved of more signal service. It should be injected pretty deeply at the seat of pain, but never into the nerve. One twelfth of a grain may be first used, increased rapidly to a third or half a grain. It relieves pain, although not in the same degree as morphia, but it has a powerful action in promoting the subsidence of the inflammation, as described in the account of neuritis. Morphia, although equally effective elsewhere, may also be injected with advantage over the inflamed part of the nerve, so as to combine the counter-irritation of cutaneous acupuncture with some local as well as general sedative influence. Simple acupuncture along the course of the nerve has been recommended; it gives temporary relief to the sciatic pain, as does any superficial pain, but the cases are very few in which it has a permanent effect. Sedative or counter-irritant liniments and ointments may also be applied along the course of the nerve; the most useful are belladonna liniment mixed with an equal part of chloroform liniment, and aconite ointment, rubbed in until distinct tingling is produced. Electricity is chiefly useful in the later stages; its method of use has been described in the account of the treatment of neuritis. In very obstinate cases, nerve-stretching has done good; sometimes, perhaps, by releasing the nerve from compressing adhesions, but probably more often by effecting an energetic counter-irritation, and enforcing a beneficial rest.

To prevent recurrence the causes should be carefully avoided, and any gouty state lessened by an appropriate regimen. Slight threatening pain should be met by more rest, counter-irritation by sinapisms, and the careful avoidance of sudden movements especially. If there is the sacro-lumbago that often precedes sciatica, the pain produced by rising from a seat may often be lessened by a few moments' rubbing.

MULTIPLE NEURITIS.

The term "multiple neuritis," or "polyneuritis," is applied to the condition in which many nerves are inflamed simultaneously or in rapid succession. This multiplicity is its most obtrusive feature. In most forms, moreover, the nerves affected are the same on the two sides; the neuritis is not only multiple, it is symmetrical; and when, as is sometimes the case, only a few nerves are involved, even one only on each side, the symmetry becomes a more salient feature than the multiplicity. Another important characteristic is its peripheral distribution; usually most intense at the extremities of the nerves, it lessens progressively towards the centre, commonly ceasing long before the nerve-roots are reached. Hence it has also been termed "peripheral neuritis," a name that is accurate but somewhat confusing, inasmuch as the term has been applied to affections of nerve-trunks generally, in distinction from central diseases causing similar symptoms.

The discovery that certain combinations of symptoms, formerly thought to depend on disease of the spinal cord, are really due to disease of the peripheral nerves, is one of the most important steps in the recent advance of pathology. It has profoundly modified many of our conceptions, not only of the processes of disease, but of the range and action of certain morbid influences, and has shown that much of our supposed knowledge of the central affections of corresponding aspect was erroneous, and the history of these maladies has to be reinvestigated.

The occurrence of multiple neuritis was first demonstrated by Duménil, of Rouen (1864), although the leprous form had been previously described by Virchow. Graves, indeed, long ago suspected that many cases of paralysis were due to disease of the nerves, but he based his opinion on the normal aspect of the spinal cord; and in most of the cases he describes it is probable that modern methods of examination would have revealed disease. Naturally, also, symptoms so peculiar had attracted the notice of clinical observers, and were described from time to time; first, perhaps (1822), by Dr. J. Jackson, of Boston, U.S.A., and, much later, but fully, by Duchenne (1858).* Duménil's observations attracted little notice, and it was not until fresh facts were brought forward by Joffroy (1879), Leyden (1880), and Grainger Stewart (1881), that attention was generally directed to the subject. During the last ten years a very large number of observations have been published, and the general history of the disease is now established on an adequate basis.

The peculiarities of distribution of multiple neuritis harmonise with what is known of its pathology. In isolated neuritis the primary affection is of the nerve-sheath and connective tissue—the neuritis is "adventitial;" in the symmetrical multiple forms the nerve-fibres themselves are almost always the seat of the primary process; the connective tissue and sheath suffer secondarily and in proportion to the acuteness of the process in the nerve-elements—the neuritis is "parenchymatous."

The forms of multiple neuritis which are thus characterised by their symmetry and parenchymatous nature are due entirely to a morbid blood-state having a direct influence on the nerve-tissue. Their cause is the presence in the blood of some virus, often an organic or inorganic chemical compound, to which the nerve-fibres are susceptible, just as they are susceptible to curara, or the motor-cells to strychnia, or the nerves for accommodation to atropia. It is an instance of what has been termed "selective action," the manifestation equally of a peculiarity in the acting virus and in the structures acted on. The peripheral distribution of the affection is probably due to the fact that the vital and nutritional energy of the nerve-fibres lessens with the distance from the cells of which they are part, and from which their vitality is derived. The relation to a blood-state as the cause explains also the symmetry of the affection, since the like structures on each side possess the same characteristics, and, being equally exposed to the morbid influence, there is no reason why the nerve-elements on one side should suffer alone, or even in less degree, than those on the other side.

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* The first ascription of a form of paralysis to this lesion was by Dr. Todd, in the case of lead palsy. "I believe that the muscles and nerves are early affected, and that at a later period the nerve-centres become implicated. The nervous system is thus first affected at its periphery, in the nerves, and, the poisoning influence continuing, the contamination gradually advances towards the centre" ('Clin. Lect.,' 1854, p. 9).

Isolated neuritis may be caused in part by a constitutional or blood state, but is not due to this alone; some exciting cause is in operation, acting locally and determining the position of the affection. Hence it is rare for such neuritis to be multiple, and when more than one nerve is affected the distribution is irregular and not symmetrical. Gouty neuritis affords an illustration of this. Moreover, in these forms the connective tissue, and especially the sheath of the nerve, is the part primarily affected, and the nerve-fibres are damaged only in a secondary manner. Between the two chief forms of neuritis, the isolated or irregular adventitial and the parenchymatous symmetrical forms, there is thus an essential difference.

Between the two classes, however, certain forms seem to occupy an intermediate position. These are the forms of multiple neuritis in which sometimes the one and sometimes the other element in the nerve seems to suffer most, or in which both are involved. We must, however, distinguish from these the cases in which mere acuteness of the process causes both elements to be involved; in which, for instance, a very acute parenchymatous neuritis causes some degree of adventitial inflammation, or acute adventitial inflammation involves the nerve-fibres. These cases conform in distribution to the type to which they belong; the primary parenchymatous neuritis, and it alone, being strictly symmetrical. But there are other cases in which the causes are combined. Alcoholism, for instance, the most common toxic cause, may be due to a mode of life that has also produced gout, and then isolated gouty perineuritis may accompany general polyneuritis of parenchymatous nature and symmetrical distribution. Exposure to cold, again, may cause a "rheumatic" perineuritis, either of a single nerve, or of a few nerves irregularly distributed; and it may also produce a blood-state allied to that of acute rheumatism, and thus a symmetrical parenchymatous polyneuritis. An important cause of neuritis is toxæmia due to the development of specific organisms in the blood, sometimes those that cause a definite disease, as diphtheria or smallpox. Such organisms seem occasionally to fix themselves in the connective tissue of the nerves, causing an irregular adventitial neuritis, and also to leave behind a product, perhaps a chemical substance, comparable to alcohol, capable of causing a parenchymatous neuritis. The two forms may then be conjoined in a puzzling manner. It is probably thus that the forms of diffuse or total neuritis are produced, occasionally met with in diphtheritic paralysis.

From these considerations it follows that symmetrical multiplicity means a blood-state acting alone; irregular multiplicity generally means a constitutional state combined with local determining causes. When very few nerves are affected irregularly, the general influence sinks to the level of a predisposition, and this is not always to be recognised when a single nerve is affected.

The classes into which multiple neuritis may be divided can now be better understood. They depend chiefly on the several causes to which the affection is due. The acuteness or slowness of its onset has also been made a ground of distinction. As we have seen, the less acute the process the more perfect is the limitation to the one element, the sheath or the nerve-fibres. In the most chronic form the process in the nerve-elements resembles a degeneration, and has been termed "degenerative neuritis." Underlying this distinction, however, was the idea that only the chronic forms commence in the nerve-fibres themselves, and that in the more acute forms the connective tissue is also primarily involved. It is certain, however, that this is not the case, although the secondary affection of the connective tissue is intense in proportion to the acuteness of the disease. From most causes we meet with cases both acute and chronic.

The forms of multiple neuritis, classified according to what is known of their causes, may be provisionally thus tabulated. No arrangement can be free from anomalies until we know more of the causes and of the mechanism by which the varieties are produced. Provisionally, however, that we may survey their relationship, the forms may be divided into five groups.

I. TOXIC: due to the presence in the blood of a known poison, which may be—
  (a) *Metallic:* lead, arsenic, silver, &c.
  (b) *Non-metallic,* as alcohol—the most frequent cause. With this may perhaps be placed the neuritis that occurs in diabetes, which probably depends on the presence in the blood of some substance allied to sugar, and formed only during the altered metabolism of this disease.

II. TOXÆMIC: due to some virus in the blood, the precise nature of which is unknown, but which is probably in most cases either an organism or a chemical product of the growth of organisms. These cases may be divided into two classes.
  (a) *Primary,* in which the neuritis is part of the first effects of the virus, as distinguished from the cases in which this first produces some other disease. The primary forms again may be

subdivided, according as the virus is derived from without, or (apparently) generated within the body.

(1) Extraneous, the virus being derived from without, as are the organisms that cause directly the neuritis of leprosy; and probably also some forms of neuritis the origin of which has not yet been based.

(2) Intrinsic, in which the virus is produced within the body, as probably in many cases of septicæmic neuritis after wounds, &c. It is possible, however, that these cases may ultimately be found to have an extraneous origin.

(*b*) *Secondary*, in which the toxæmic agent first causes some definite disease, which is followed by polyneuritis as a sequel, an interval of some weeks or even months elapsing between the two effects. The neuritis is then sometimes said to be caused by the disease. Probably, as already explained, the neuritis is generally due to some chemical product of the growth of the organisms in the body. Examples of this are the polyneuritis that follows diphtheria, smallpox, variola, typhoid, tubercle, and perhaps syphilis. In some of these, as already explained (p. 110), two mechanisms may be at work—one direct, the other indirect.

III. ENDEMIC: the neuritis that is due to local organisms which have this for their primary or secondary effect. The chief forms are malarial neuritis and the disease called beri-beri. Strictly, however, these belong to II, *a*, 1, primary toxæmic neuritis due to a cause acquired from without; but their peculiar local characteristics make it convenient to separate them.

IV. RHEUMATIC: the multiple neuritis that follows exposure to cold, and is probably due to a morbid blood-state like that which gives rise to rheumatic fever, pneumonia, polio-myelitis, and the like. It is possible that these belong strictly to II, *a*, 2, the primary neuritis due to a blood-state of intrinsic origin, but they constitute a well-marked group.

V. CACHECTIC and SENILE forms, in which degeneration of the nerves seems part of general mal-nutrition, and begins at the periphery where their vitality is lowest. Some of these cases may be really neuropathic, and due to the same causes as produce degeneration in the spinal cord; while among the senile forms must be reckoned not only those due simply to age-failure of nutrition, but the rare instances in which arterial degeneration and lessened blood-supply produce local necrotic changes in the nerve.

It is not necessary to consider here the particulars regarding the causation of all these forms. The neuritis due to the Metallic Poisons is described in Vol. II in connexion with their other effects on the nervous system; there also Diphtheritic Neuritis is considered in the account of this paralysis (which is not wholly peripheral), and most of the secondary toxæmic forms find their place in the account of the Paralyses after Acute Diseases. Malarial neuritis, Beri-beri, and Leprous neuritis are considered separately at the end of this section. The forms that need special consideration in this place are certain varieties of "primary" polyneuritis, toxic or toxæmic, the "secondary" toxæmic forms of intrinsic origin, the rheumatic, diabetic, and the cachectic forms—in most of which the symptoms of polyneuritis develop as an apparently independent disease, and not as the sequel to some other affection. Of the other forms, however, some notice must be taken where they come into important contrast or comparison with those now specially considered.

By far the most common cause of multiple neuritis is alcoholism, and this variety must therefore furnish us with the most important facts regarding the clinical history of the disorder. But it may be well first to consider briefly the causation of some of the less frequent varieties.

Syphilitic neuritis is mainly an affection of single nerves, and, as such, has been already considered. These probably suffer in consequence of the direct action of the organisms on the adventitial tissues, and coincide in time with other "secondary" syphilitic lesions. It has been conjectured that true acute multiple neuritis may be directly syphilitic, but the evidence of this is insufficient. We may, however, see a true parenchymatous syphilitic polyneuritis in the tabetic form, in which the sensory fibres are affected at the periphery; tabes is generally a post-syphilitic disorder, and parenchymatous degenerative neuritis is a common, and sometimes the only lesion in this affection. This "peripheral neuro-tabes," as it is termed, may not unreasonably be regarded as an effect of some product of the specific organisms, comparable to the less chronic forms of neuritis that succeed less chronic specific diseases, such as diphtheria or smallpox. The subject is considered in the section on Locomotor Ataxy, but it is important to mention it here, because this disease presents an example of central and peripheral lesions due to the same cause, and because an affection of the sensory nerves almost identical in distribution and nature, and giving rise to similar effects, is sometimes the consequence of alcohol, and occasionally also of other agents, as arsenic.

Tubercular polyneuritis is a form regarding which we have still much to learn. It has occasionally developed in the course of phthisis, and has been found to be of the purely parenchymatous or degenerative variety. Severe cases, with characteristic motor and sensory symptoms, in which other causes, such as alcohol, can be excluded, are rare, but their occurrence is well established,* and they seem to justify the conclusion that the tubercle bacillus is capable of producing a poison having a specific action on the nerves—a mechanism also indicated by the fact that the bacilli themselves cannot be found in the affected structures. It is said that similar slighter neuritis is frequently to be found after death in cases of phthisis, and probably produces symptoms that are overlooked in the general prostration of the later stages of the disease. Some severe cases recorded as tubercular are, however, not free from the suspicion that alcohol was in part or wholly the cause of the neuritis. There is, moreover, another relation between multiple neuritis and phthisis; the latter often develops in the course of alcoholic neuritis, and is a frequent cause of death. It is not improbable that a neuritis of the pulmonary branches of the vagus leads to the low pneumonic form of phthisis that is generally found in such cases, and this may even render the tubercular organisms more effective if they find entrance into the lungs. Although gout is so powerful a cause of isolated neuritis, there is no evidence that it causes the multiple form, although it may perhaps modify the symptoms of this by adding to it an isolated perineuritis.

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* See Oppenheim, 'Zeitschr. f. klin. Med.,' 1886, p. 230.

The Acute Diseases that may be followed by neuritis are especially diphtheria, variola, measles, typhus and typhoid fevers. The interval that usually elapses between the acute disease and the neuritis supports the opinion that the latter is due to a chemical poison produced by the specific organisms; and so also does the perfect symmetry of the symptoms, since the toxic agent must be such as can have an equal action on all similar structures. Moreover, the occurrence of the neuritis is not related to the severity of the primary disease, and thus seems to be due rather to some peculiarity in the special organisms, such as so often determines peculiar features in an epidemic. In one case, which developed six weeks after varioloid, with pains in the limbs, atrophy of the arms and legs, &c., the purely neuritic nature of the morbid process was ascertained six months later, when the patient died from pneumonia. In diphtheria, as already pointed out, we seem to trace a double mechanism,—an early direct action of the organisms on the adventitial tissue in some cases, and a later parenchymatous neuritis due to some product of their growth, more abundant and effective in some cases than in others. Malarial neuritis seems to occupy a position intermediate between the secondary and primary forms, since it sometimes follows malarial fever, and sometimes develops without previous fever in those who have been exposed to the influence of malaria. It is described separately as one of the endemic forms.

Of the *Primary Toxæmic varieties* a typical example is presented by another endemic form, beri-beri, which is the sole result of an organised virus, or of a product of the organisms. This also is considered in a separate section, but its history deserves careful study on account of the light it throws on the pathology and causation of other forms of polyneuritis, in which the processes are less simple and therefore less clear.

An instructive contrast to beri-beri, which is a purely parenchymatous form, is presented by leprous neuritis, also described separately. Here we find a purely adventitial form, due to the direct presence of the organisms in the connective tissue. The contrast presented by these two forms is almost complete.

Septicæmic polyneuritis follows some wound or injury or local inflammation capable of giving rise to blood-poisoning. In some cases it has been unaccompanied by the usual symptoms of septic blood-states, but has followed its cause at an interval that suggests, taken with other analogous facts, that it is due to some virus produced probably by the presence of septic organisms of a special character. For instance, a man, thirteen days after a stab-wound beneath the clavicle, which healed well, had an attack of parotitis with facial palsy. On the fortieth day there developed paralysis of tongue, vocal cords, and limbs, and on the sixth day after the onset of these symptoms he died from respiratory palsy. Extensive peripheral neuritis was the only nerve lesion.*

In a less severe case recorded by Barr† the symptoms commenced three weeks after a neglected wound of the hand, which produced inflammation of the lymphatics, extending up the arm to the axilla. The symptoms of neuritis began in the injured limb, and invaded successively the other extremities, causing characteristic weakness in the lower legs, with loss of the knee-jerk. He slowly recovered. It is possible that some cases of this class result from unsuspected foci of inflammation within the body, in which pus becomes pent up and causes the blood-state.

In such cases as those due to a possible internal source of blood-poisoning, multiple neuritis develops as a disease apparently primary without obvious cause if the source of the septicæmia has been undiscovered. Other primary cases occur, however, in which there is no cause for septicæmia, and the only explanation we can give of these is that they are probably due to a virus received from without, the precise nature of which is unknown. I have met with some cases in which the symptoms of multiple neuritis, perfectly characteristic, came on without any discoverable cause, and ran a course in most cases mild, and ending in recovery; in one or two others severe, and ending in death. Some cases of what is popularly termed "blood-poisoning" are probably of this nature. If certain drain poisons, probably organismal, are capable of giving rise to a disease, diphtheria, of which multiple neuritis is often part, there is no improbability in some forms of virus of similar character and analogous source, having such neuritis for their chief effect. In this connection it is instructive to note that an epidemic is on record in which paralysis preceded the throat affection (see 'Diphtheritic Paralysis,' vol. ii). As an instance of this primary form may be mentioned the case of a medical practitioner who was attacked with obstinate vomiting and diarrhœa succeeded by constipation, for which no cause could be discovered, quickly followed by "numbness" in the hands and feet, and a difficulty in using them. When I saw him, a fortnight later, there was loss of tactile sensibility, limited to the palms and soles, weakness of the legs, and no knee-jerk. The weakness rapidly increased and extended, and at the end of five weeks from the onset he died. Such a case presents all the features of the toxic form.‡

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* Roth, 'Corr.-Bl. f. Schw. Aerzte,' 1883, No. 13.

† 'Amer. Journ. Med. Science,' Feb., 1889.

‡ It should be mentioned that other circumstances excited a suspicion of intentional chronic poisoning. This fact does not lessen the suggestiveness of the case. Multiple neuritis must henceforth constitute an element in many questions of medical jurisprudence. Its discovery might give greater weight to indications of some forms of poisoning.

The *Rheumatic* form embraces all the cases of polyneuritis that are due to exposure to cold. In the typical cases of this class the general distribution of the affection and other features indicate that a blood-state must be the immediate mechanism by which it is produced. We know that rheumatic fever, due to a similar exposure to cold, must depend on such a morbid blood-state, produced in some way by the disturbing influence of the chill on the chemical or other processes within the body. We know also that the rheumatic poison thus generated must vary much in its precise nature by the great variations in the acute rheumatic lesions that it causes (see 'Chorea,' vol. ii). The same exposure, in another person, may give rise to pneumonia. Hence it is not surprising that the effect of the exposure should, in some persons, be such as to cause a blood-state capable of producing multiple neuritis, symmetrical and parenchymatous, especially when we know that this may actually be accompanied by acute pneumonia, and by other evidence of profound toxic state of the blood. In a case recorded by Putnam, fatal in seven days from paralysis of the respiratory muscles, and distinctly due to cold, there was swelling of the spleen such as is met with in typhoid and other acute toxæmic states. But exposure to cold has also the power of causing an isolated neuritis (*e. g.* of the facial or the sciatic nerve), a neuritis adventitial in nature, essentially a perineuritis. Nor is it surprising that such an adventitial neuritis should occasionally involve more than one nerve—should, in fact, be a polyneuritis. The evidence that such polyneuritis may be produced by cold is given in the chapter on Acute Atrophic Paralysis (Polio myelitis), with which it may coincide, as a result of the same exposure. The connective tissue of nerves is of the same nature as that on which the rheumatic poison exerts its special influence, and the modes of onset of sciatica have shown us that the rheumatic process may pass from the fasciæ to the nerve-sheaths. A consideration of these and other facts makes it probable that cold may cause (1) an isolated adventitial neuritis; (2) a multiple neuritis of the same character, irregular in distribution, probably by the agency of a blood-poison related to that which causes muscular rheumatism and the affection of the fibrous tissues of the joints in rheumatic fever; (3) a symmetrical parenchymatous polyneuritis due probably to a special blood-state of greater specificity than that which produces the adventitial form, and perhaps allied to that which gives rise to pneumonia and to cerebro-spinal meningitis. Pneumonia, at least, may be combined with multiple neuritis as a result of the same exposure.

The *diabetic* form presents many varieties, and our knowledge of it is still imperfect. It is supposed that inflammation of single nerves frequently underlies the obstinate forms of neuralgia met with in this affection; but besides this a symmetrical multiple neuritis, parenchymatous in nature, is occasionally met with, and is said to present all the varieties that are produced by alcohol, and of various degrees of severity. It is not, however, related to the amount of sugar in the urine—in many of the sufferers this has been small (in one, indeed, as low as one half per cent.); nor is it readily influenced by a dietetic reduction of the amount of sugar in the blood. Hence it is supposed to be due to some toxic product that is formed in the perverted metabolism of the diabetic, comparable to acetone, although certainly not acetone itself. The fact that the reduction of the amount of sugar formed has little influence on the condition suggests that the poison is not a product of the decomposition of sugar, but rather a material formed in the place of sugar by some modification of the chemical processes that lead to the increased production of the latter. This form is of special interest on account of the close chemical relation between sugar and the most potent cause of multiple neuritis, alcohol.

Alcoholic polyneuritis is the form most frequently met with, at any rate so far as well-marked cases are concerned; this form preponderates in most countries over all others put together, but at present many of the slighter sensory forms escape recognition, and the preponderance will probably be much less when all cases are recognised. It results chiefly from the stronger forms of alcoholic drinks, and especially from spirit-drinking. It is more common among those who take small quantities frequently than among those who indulge in an occasional "bout" of intemperance, probably because the total quantity consumed is greater by the former. It is far more frequent among women than among men—probably three times as frequent. How far this is due to a special predisposition on the part of women, or to the conditions under which it is taken, is yet doubtful. On the one hand continuous drinking is more common among women than is occasional intoxication; and women who take alcohol to excess generally do so in the form of spirits, while men often take it in the form of beer. At the same time the opposite proclivity is seen in the case of delirium tremens, and it would seem as if there were a difference in the nervous system of the two sexes, whereby a susceptibility of the nerves of women replaces that of the brain in men.

The amount of alcohol habitually taken, and the duration of the habit before the onset of the affection, vary much. It has been thought that a neuropathic disposition renders a less quantity and briefer period adequate. Other causes often co-operate with alcohol in exciting the disease, especially exposure to cold, and, in the lower classes, insufficient nourishment. Exposure is especially effective in those in whom slight symptoms exist but attract little attention, and the condition of the nervous system is, as it were, prepared for a severe outbreak.

Cachectic and Senile Forms.—Regarding these little remains to be added to what has been stated at p. 112. The evidence rests chiefly on the post-mortem discovery of the changes in the nerves, but it is probable that more careful attention to the point will show that symptoms are not unfrequent which now receive little attention, or are regarded as functional only. More attention is also needed by the cases in which a neuropathic tendency or some depressing emotion seems to be the cause of this disease, as they may be of the corresponding degeneration of the pyramidal fibres within the cord in "lateral sclerosis," a degeneration which also begins at the extremity of the fibres and extends upwards.

Atheromatous Neuritis, already mentioned as a variety of the senile form, merits separate recognition on account of its peculiar mechanism. In these cases the nerves of the limbs have been extensively damaged through atheroma (or "arteritis obliterans") of their arteries. Necrotic inflammatory processes take place in the parts of the nerves supplied by the affected vessels, and there result symptoms that resemble those of the toxic form in character, although differing in the greater irregularity of distribution. The symmetry that constitutes so striking a feature of the common cases is absent, but arterial degeneration may be symmetrical, and an incomplete rough symmetry may thus result. It is conjectured that such disease of the nerves will be found to be frequent in the cases in which senile disease of the arteries gives rise to gangrene, and that it is partly through the agency of the neuritis that the gangrene is produced; but this view overlooks the potent influence of the arterial obstruction itself.

GENERAL ETIOLOGY.—Multiple neuritis is a disease of adult life. The only form met with in children is that which sometimes accompanies polio-myelitis, and may perhaps now and then be met with apart from the spinal malady, as an infantile variety of multiple adventitial neuritis, irregular in distribution. The other common forms occur chiefly between twenty and fifty; the alcoholic between thirty and forty twice as frequently as in either the previous or later decade, but it may be met with up to sixty years of age; while the rare senile (purely degenerative) variety, and the still more rare arteritic form are met with at still later ages. Females constitute a majority of the cases, the degree of which cannot yet be estimated; it is due solely to their liability to alcoholic neuritis, in which they amount to at least 70 per cent. The rheumatic and toxæmic forms, on the other hand, occur more frequently in males, probably from their greater exposure to the causes of the disease.

More than one cause may co-operate in producing the affection, and then we may distinguish the one as "predisposing," the other as "exciting," although, as a rule, either would be adequate alone were its degree more intense. The double causation can most often be traced when cold or some depressing influence co-operates with alcohol, or when the influence of the latter is conjoined with that of tubercle, as either the primary or secondary cause. Even more frequently the symptoms follow some cause of constitutional depression, inadequate alone. In one case this was an attack of acute gastric catarrh with vomiting, the result of the alcoholism which was the real cause of the neuritis. In other cases a depressing emotion has seemed to be the excitant, and, as we have seen, this appears to be sometimes alone sufficient. In one alcoholic case the injurious effect of painful emotion (due to the death of a son) was at once to arrest improvement, and to excite a progressive increase in the symptoms, which continued until death a few months later. Anæmia probably acts chiefly as a predisponent, lowering the nutrition and resisting power of all the tissues, and rendering a slighter cause effective. Thus, in the case of an anæmic girl recorded by Barrs, the symptoms followed immediately on bathing the feet in cold water, when heated.

SYMPTOMS.—The symptoms produced by multiple neuritis differ widely, as has been already intimated, according to the character of the affection of the nerves. They are of three classes—motor weakness, sensory disturbance, and inco-ordination. The motor weakness depends on an affection of the motor nerve-fibres, and usually involves first and chiefly the flexors of the ankle and extensors of the toes, and the extensors of the wrist and fingers in the forearm, muscles that are homologous and supplied by corresponding nerves, the anterior tibial or peroneal nerve in the leg, the radial branch of the musculo-spiral in the arm. The result is the characteristic "wrist-drop" and "foot-drop," shown in Fig. 58. Other muscles suffer in severe cases. The sensory symptoms consist of subjective sensations of tingling, &c.; pains of various character, seat, and degree, often referred to the nerves or deep parts; tenderness of the skin, nerve-trunks, and muscles, and loss of cutaneous sensibility. The inco-ordination resembles that present in the slighter degrees of locomotor ataxy, and is often associated with loss of the muscular sense. These symptoms will be considered in detail. According to the predominance of one or another of the three sets, corresponding varieties are distinguished,—(1) a *motor* form, in which there is loss of power with or without the other symptoms; (2) a *sensory* form, in which there is no marked muscular weakness or inco-ordination, but in which sensory disturbance—irritation or loss—is the chief symptom; (3) an *ataxic* form, in which, without marked loss of power, with or without sensory symptoms, inco-ordination is the dominant symptom.

As mentioned in the introductory section, the varieties of polyneuritis that are due to special metallic poisons and to certain acute specific diseases are described separately, in the second volume of this work, among the general diseases of the nervous system. The common alcoholic form is alone suitable to convey a knowledge of the general features of the affection, but the account of its symptoms must be completed by some mention of those of the less common forms, which do not need separate description elsewhere.

It should be clearly understood at the outset that no set of symptoms is exclusively related to a single cause. The various forms which depend on the incidence of the disease on the motor or the sensory nerves in preponderant degree, are met with in consequence of each of the common causes, although, as we shall see, some causes give rise more frequently to one form than to another.

The onset and course of multiple neuritis vary much in different cases, both of the same class and of the several classes. Premonitory symptoms occur in some cases. They have been observed chiefly in the alcoholic form, but occur in others also. When due to cold or other causes of constitutional disturbance, the special symptoms of the general state may immediately precede the onset. Thus, after exposure to cold, general rheumatic symptoms, with fever, bronchitis, and other signs of catarrh, may immediately be followed by the symptoms of neuritis. The symptoms that have been regarded as premonitory are especially "numbness" and tingling of the fingers and toes, or of the palms and soles, or the lower parts of the limbs, vaso-motor disturbance in the extremities (as pallor of the fingers), and painful cramp in the calves or elsewhere, together with dull rheumatoid pains. These constitute the first symptoms of the disease, but when they exist alone for months before the onset, they may be regarded as "premonitory." All these symptoms are probably due to the influence of alcohol on the nerves, disturbing the function of the structures whose nutrition undergoes subsequently a corresponding impairment.

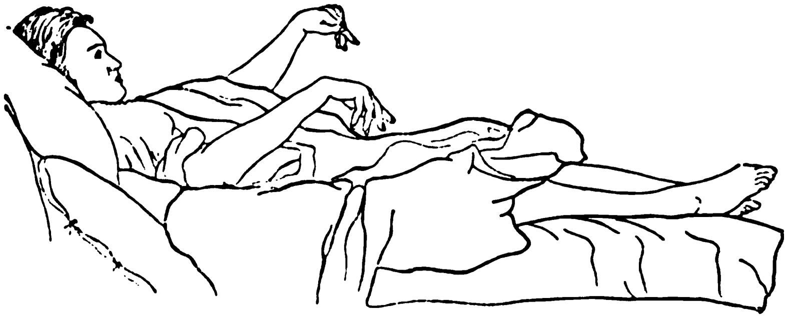

FIG. 58.—Multiple alcoholic neuritis; palsy of extensors of wrist and flexors of ankle. (From a photograph by Mr. Hyde Marriott, B.Sc.)

The onset itself may be acute or subchronic, and is sometimes so slow as to deserve the name of chronic, especially in the slighter sensory form. An acute onset is on the whole rare in alcoholic cases unless the malady is "excited" by some coöperant influence. Thus, in one alcoholic, a prostrating attack of vomiting, lasting for ten days, was followed by an onset of the symptoms of neuritis so acute that at the end of a week the patient was unable to stand, and there was general impairment of sensibility. It is met with chiefly in cases due to cold or toxæmic states; in these it may be attended by severe constitutional disturbance, rigors, and considerable pyrexia. In other forms the elevation of temperature is usually slight or moderate, but sometimes amounts to 3° or 4°, and may last from one to three or four weeks. The first definite symptom (if it has not existed before) is often the tingling, or "pins and needles" in the extremities, already mentioned, with vague "rheumatic" pains, which become more acute. Motor symptoms are usually soon added, palsy or inco-ordination, or both. They are seldom absent in acute cases. The pronounced symptoms of the disease may resemble in character those produced by the inflammation of single nerves, differing only in distribution, and involving all the functions of the affected nerves; this is often the case when the malady is acute in onset. But when chronic, as already stated, the fibres may be affected according to their specific function, and to this the symptoms are then limited, so that they resemble those that are ascribed to a disturbance of special elements of the spinal cord, rather than the affection of isolated nerves.

The motor form involves either the upper or lower limbs or both, but always the corresponding limbs on both sides, and their distal extremities, the hands or the feet, first and chiefly.* Whichever limbs, arms or legs, present the first symptoms, suffer in greater degree throughout, and it may be alone. The feet suffer more frequently than the hands; motor symptoms may exist in the legs, and only sensory symptoms in the hands, and this when the weakness in the legs is extreme. In whatever part the affection occurs various sensory disturbances, and especially pains, commonly precede the weakness, and increase with it. They are accompanied by tenderness generally along the nerve-trunks, but almost invariably in the muscles, and sometimes of the skin. These pains may occur in the limbs that do not become paralysed if the action of the cause can be arrested. Although loss of power is the obtrusive feature, some loss of co-ordination generally accompanies it, and is often the means by which the attention of the patient is first directed to the commencing disability. He finds a difficulty in balance when standing, or in performing the finer movements with his fingers; and his attention being thus drawn to the part, he finds, on further investigation, that there is some actual defect in the power of extending the wrist and fingers, or in raising the toes or foot from the ground during the forward movement of the foot in walking. Tremor is often conspicuous in alcoholic cases long before the loss of power. Whenever there is weakness in the leg the knee-jerk can no longer be obtained, probably because the afferent muscle-nerves suffer more widely than the motor nerves. The constancy of this loss is very great, and it is not related to any one class of symptoms, but occurs equally in the paralytic and ataxic forms of neuritis. At the same time it is not absolute; the knee-jerk seems to persist in very rare cases of slight degree, as mentioned on p. 126.

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* The very various symptoms of the disease are well illustrated by an instructive series of cases published by Dr. Buzzard, 'Paralysis from Peripheral Neuritis,' 1886.

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When the first symptoms are in the legs the first motor loss is the inability to raise the toes from the ground in walking, owing to imperfect flexion of the ankle-joint and extension of the toes. But this symptom may be to a large extent obscured by the interference with movement occasioned by the tenderness of the muscles and the hyperæsthesia of the skin, which commonly coexist and prevent the exertion of the amount of power that is possessed. There is a tendency for the nerves of the sole and the palm of the hand to be especially involved in the hyperæsthesia—in obedience to a law of pathological susceptibility which is to be traced not unfrequently in polyneuritis, especially in the tabetic, diphtheritic, and toxæmic forms.

These effects of the sensory irritation may render it difficult to determine the common course of the motor weakness, the order in which the muscles suffer, and the relative degree of their impairment at different periods. The conspicuous fact is that the muscles first to suffer are those supplied from the external popliteal nerve (anterior tibial group), and these are followed by other muscles below the knee, except in the very rare instances in which palsy does not spread beyond the muscles first affected. The muscles above the knee suffer later and less, and more frequently escape considerable impairment. The flexors of the knee, although supplied by the sciatic nerve, do not suffer more than the extensors, or more frequently, so far as the tenderness permits us to judge. Least frequently and in least degree the muscles suffer that move the hip-joint; it is only in the most severe cases that these are appreciably affected. Very rarely is the innervation of the bladder or rectum involved in the disturbance of polyneuritis. Interference with these functions may generally be regarded as evidence that the morbid changes are not limited to the nerves, but involve the spinal cord itself.

In the arms, the first and chief defect in power is in the extensors of the wrist and fingers. Both arms are affected together or in quick succession—one never suffers alone in considerable degree. Thus the palsy resembles at first, and sometimes throughout, that which is familiar as the effect of lead poisoning. The resemblance may extend to the escape of the extensor of the metacarpal bone of the thumb, and of the supinator longus. Lead palsy, indeed, is an example of peripheral neuritis of the "toxic form," and differs from the general form in the limitation of even severe palsy. It seems that the radial nerve (of the musculo-spiral) is that which, in the upper limbs, is the most prone to suffer—a fact which is at present unexplained. It probably depends not on any peculiarity of position, but on some deeper functional relation and position among the nerves of the body, since it obtains when the affection is of the peripheral extremities of the fibres and does not involve all the fibres of the nerve. The radial nerve is homologous with that in the leg of which the fibres are the first to suffer—the peroneal,—regarding which the same facts are true.

It is only in the cases due to metallic poisons that the palsy becomes considerable without spreading to other muscles. Generally the flexors of the wrist and fingers become weak, and so do the interossei, while the last to suffer are the thenar and hypothenar muscles. It is rare for the palsy of any muscles except the extensors to be complete. In more severe degree the muscles above the elbow suffer, last and least those of the shoulder. The muscles below the elbow may be almost powerless when those above it are scarcely affected.

In severe cases the trunk muscles are involved—the diaphragm, and the muscles of the thorax and abdomen. Less rarely, but also only in severe forms, increased frequency of the pulse and palsy of the vocal cords indicate that the fibres of the vagus are suffering. Very seldom the facial muscles, or those of the tongue, are involved. Affection of the motor ocular nerves, of the pupils, and nystagmus, have been met with only in the most severe cases, and chiefly in those due to toxæmia. The nerves of the pharynx and alimentary viscera seem always to escape. It is more doubtful whether this is true of the branches of the lung.

Thus the stress of the affection falls on the muscles of the extremities, and those that are affected first also suffer alone in slight cases, and most in those that are severe. As they become weak, and sometimes even earlier, they become tender, and this muscular tenderness usually increases to an extreme degree, and becomes even more obtrusive, in many cases, than the tenderness of the nerves. The limb cannot be grasped, even gently, without cries of pain being elicited, and every change of posture gives rise to distress. This is an important sign, and is no doubt due to the fact that all the nerves of the muscle suffer, the afferent as well as the motor twigs. Very rarely it is absent. It increases the pain of an electrical examination, and, after a strong current has been applied, the muscles may ache for hours—a fact that should inculcate scrupulous consideration in all such procedures.

The affected muscles quickly become flabby and waste, and their prominences disappear. The muscular atrophy becomes extreme in some instances, but the aspect of the limbs differs from that in progressive muscular atrophy on account of the partial distribution of the affection, and because it is only in a few muscles, such as the extensors of the fingers and the interossei, that the wasting is as great as in that affection. Changes in the electrical excitability of the muscles concur with these symptoms; they present the reaction characteristic of nerve lesions, described at p. 54. The faradic irritability is lost, and that to voltaism is increased in amount, and often but not always altered in quality, in the usual manner. In slight cases, however, increase in voltaic irritability may be trifling, and may be the chief change. In the nerves the irritability to both currents lessens and is ultimately lost. If a case is examined in the very early stage, the increased irritability mentioned at p. 58 may be found, and afterwards replaced by diminution and loss. In the most severe cases the muscles most affected may quickly lose all irritability, on account of the intensity of the secondary changes in the muscular tissue, which destroy instead of merely changing their special structure and characteristic functions. Occasionally the wasting is concealed by œdema, but now and then (as I have seen) the size of the muscles may be little reduced even when they are totally paralysed, with extinction of faradic and great reduction of voltaic irritability. But the wasting is only deferred, and extreme atrophy ultimately occurs. In such cases there is probably a rapid fatty degeneration of the muscles, and the fat accumulates between the fibres so as to maintain, for a time, the bulk of the muscle. An extremely rare alteration is the preservation of some voltaic irritability in the nerves as well as in the muscles, faradic irritability being lost in both.

As in other cases of unequal palsy, the less affected muscles of the limbs are apt to undergo contracture and shortening, especially when the palsy causes the part to assume habitually a certain position under the influence of gravitation, or a certain posture is adopted by the patient in order to lessen pain. These contractures occur chiefly in the lower limbs. The foot-drop shown in Fig. 58 is especially frequent, from the palsy of the flexors permitting the foot to fall as the patient lies in bed, under the influence of gravitation and of the unopposed extensors. The effect of posture is seen in the contraction of the flexors of the knee, which may be fixed at a right angle, and to a less degree of those of the hip, on account of habitual flexion of these joints to escape the pain occasioned when the parts are put on the stretch in the extension of the limb.

The affection of the sensory nerves of the skin is sometimes absent, more often it is conjoined with that of the motor nerves, and sometimes only with that of the sensory muscle-nerves. It very seldom exists alone. Its first manifestation is the tingling and other subjective sensations already mentioned, to which is soon added the hyperæsthesia which is often so troublesome throughout the disease. Sensory disturbance is usually more extensive than the motor palsy, but is greatest at the most affected parts, the extremities of the limbs. Except in slight cases, the damage to the sensory fibres becomes sufficient to lessen the sensibility of the skin to touch. A faint touch may be unfelt while over-sensitiveness to pain continues, and is even so intense that a strong touch may cause distress. Rarely, as I have twice seen, sensibility to pain is lost, that to touch being preserved. The temperature sense is usually normal. These sensory changes are first developed on the extremities, sometimes on the palms and soles; they may even be still more local, and only considerable on the tips of the fingers, or the hyperæsthesia may be chiefly under the nails. Spontaneous pains accompany the hyperæsthesia, dull, or acute, or "burning" in character, referred to the deeper parts of the limb, or the nerve-trunks, or the joints, and increased or excited by any attempt at movement. The pain referred to the joints may be really in them, especially in the gouty, or it may be in the nerves that pass by the joints and are disturbed mechanically by their movement. The sensory change increases in area as the disease develops, and usually extends first up the outer side of the leg and the radial side of the forearm. Although sensibility to pain is seldom lost, the conduction of pain is delayed in some cases.

In the ataxic form, inco-ordination is the chief symptom, and it may accompany the onset of muscular weakness in the motor form. It is usually accompanied by muscular tenderness, and exists with or without the affection of cutaneous sensibility just described. It probably depends on changes in the afferent muscle-nerves, the impressions from which are chiefly concerned in the process of co-ordination (see Spinal Cord, Functions). The implication of these nerves is shown by the muscular tenderness that is so marked a feature. The inco-ordination in these cases resembles closely that which exists in tabes, and the knee-jerk is lost as in that disease, while the frequent occurrence of pains in the limbs increases the resemblance. Hence the form in which this symptom is predominant has been called *pseudo-tabes*, often with the prefix *alcoholic*, from the fact that this is its most frequent cause. It has, however, also been met with from arsenical poisoning. The ataxy may involve the arms or the legs, but is most frequent in the latter. It never reaches the degree met with in advanced tabes, probably because very extensive and intense damage to the muscle-nerves only occurs when the toxic cause is intense; its effect then is not limited to a single set of nerves, but, involving the motor also, produces a loss of power, before which the inco-ordination ceases to be prominent, if it does not of necessity disappear. The general law that limitation needs chronicity of course and moderation of degree holds good in these cases on the sensory as well as on the motor side. When the inco-ordination is the prominent symptom the disturbance of sensory nerves is slight, and if there is extreme hyperæsthesia and much loss of sensation there is usually also motor palsy; and thus this ataxic form is not commonly accompanied by the intense sensory disturbance of the complete and characteristic cases. Sensory loss, chiefly to touch, is, however, occasionally met with in chronic cases of the ataxic type. The resemblance to tabes is then still greater. The distinction is considered in the section on "Diagnosis."

Tenderness of the nerve-trunks in the region of whose supply the symptoms are greatest is a very common symptom, although not always readily recognised, because they are only more tender than the adjacent structures, and the degree of difference may seem not great when all the parts are extremely sensitive. It is less frequently a marked symptom than the tenderness of the muscles, and must depend (since the pain is felt at the spot) on the involvement of the nerves of the sheath. It is only in very rare cases, chiefly of irregular adventitial neuritis, that any swelling of accessible nerves can be distinguished.

Reflex action from the skin varies much in its condition. In cases with hyperæsthesia it is often increased, even when there is considerable motor palsy; the muscles that escape cause the movement, and the impression that reaches the reflex centre is doubtless increased in degree just as is that part of it which reaches the brain and is felt as increased sensation. But in severe cases, with extensive loss of power and sensation, the skin-reflex may be lost. The loss may be observed to coincide with an increase in the other symptoms, and generally to be related to loss of sensibility to touch with extensive motor palsy; it may, however, be lost when sensation is perfect. Occasionally it is lost out of proportion to the other symptoms, motor or sensory. The muscle-reflex action (myotatic irritability), as we have already seen, is almost invariably lost. Although the knee-jerk has been observed to persist in rare cases (Déjerine), its persistence is quite exceptional, and probably depends on the escape of at least some of the fibres on which the action depends. Possibly, however, the knee-jerk is sometimes excessive in the early stages of the disease in consequence of an irritable state of the nerves on which it depends, similar to that which gives rise to the hyperæsthesia. I have more than once found it increased in cases of slight sensory neuritis (*e.g.* with symmetrical tingling and slight anæsthesia on the soles), and in one such case it disappeared as the disease progressed. On the other hand, it is often lost when there is scarcely any appreciable weakness of the extensors, although usually some tenderness of these muscles suggests an affection of the afferent path.

Trophic changes occur in prolonged cases—in the nails, skin, and hairs, and are similar to those of ordinary neuritis; glossy skin, arthritic adhesions, and thickening being the most common. Bedsores are not common—a fact well established, but not altogether easy to explain. The vaso-motor nerves may present disturbance of function, and some œdema of the limbs is common, especially in cases that are due to alcohol: the blood-state or impaired function of the kidneys probably aids in its production. It occurs about the ankles, back of the foot, and very frequently about the wrist and back of the hand. In one very severe and fatal alcoholic case the legs and back were the seat of a peculiar brawny œdema, pitting very slowly, and attended with lividity of the skin in the depending parts, which became brighter in colour on pressure. In this case endocarditis developed during the course of the malady. In one recorded case œdema was present only over the nerve-trunks in the neighbourhood of joints—a place in which the movement of the limbs especially disturbs the sheath-nerves of the trunks, to which the vaso-motor nerves of the part are probably related. Indeed, it is common for œdema to be present about the joints when it is not elsewhere, and this may be the explanation. Pain in the joints and even some effusion into them have been observed, especially in association with an acute onset, but it is perhaps more often gouty[*] than rheumatic in alcoholic cases, and its significance as to the "rheumatic" nature of the neuritis must be looked on with some suspicion. It may be occasionally a result of the disease of the nerves, since chronic changes in the joints often take place just as they do, in the inflammation of single nerves. It is especially common in the finger-joints and in the wrist, but it occurs also in the shoulder and knee, less frequently in the elbow and other joints of the legs. There is pain on movement and thickening about the joint, with ultimate limitation of movement. It is a troublesome effect of the disease, because it interferes with movement when power returns. There is much more tendency to it in some cases than in others, and it may be so extensive as permanently to cripple the sufferer.[†] The adhesions commonly re-form after they have been forcibly broken down.

The symptoms are wide-spread in proportion to the acuteness and intensity of the malady, but differ in these respects also according to its cause. The effect of metallic poisons is usually limited and confined to the arms, as in lead palsy. In alcoholism all the limbs are often affected, but the arms escape more often than the legs. From malaria the legs usually suffer alone; such cases are considered at p. 151. In cases with complex causation a general parenchymatous neuritis may be accompanied by a preponderant adventitial inflammation of some one nerve, as the facial or sciatic. The cases with most widely distributed symptoms are those that result from obscure toxæmic states, from cold, and from alcoholism. It is in these, and especially in the first, that the nerves of the heart, larynx, and of the muscles of respiration most frequently suffer. When the laryngeal nerves suffer it is usual for both adduction and abduction of the vocal cords to be weakened, seldom one movement only. The optic nerves have been affected only in rare cases with considerable blood-change. In the case mentioned on p. 126 (in which there was brawny œdema and endocarditis) there was slight optic neuritis and a diffuse œdematous retinitis.[‡] The face has also been affected on both sides in some of the cases due to exposure to cold, and also, usually in slight degree, in some toxæmic cases; it seems always to escape in cases due simply to alcohol. The sphincters almost always escape; a slight impairment of function has indeed been met with in a few very severe cases of probably pure polyneuritis, but their affection, as already stated, usually indicates an implication of the spinal cord.

The *Course* of the affection is determined by the same conditions as influence its intensity, and by the extent to which the cause is under control. The more severe and acute the symptoms, the wider is their range and the longer is their duration. Very commonly the symptoms increase during three to six weeks, then become stationary (if the cause is stopped), and after one or two months slowly improve. The first sign of improvement is a diminution in the pain and hyperæsthesia, but the tenderness of the muscles and the nerve-pains on movement often continue long after the spontaneous pains have ceased. If tingling, &c., ceased as the disease advanced their return may herald improvement. Power slowly returns, first in the muscles affected last and least, and afterwards in those paralysed and wasted in greater degree. In these the weakness lasts for many months, and shortening of the opponents of the most affected muscles is apt to occur. The contracture of the calf muscles, secondary to palsy of the flexors of the ankle, constitutes a grave hindrance to the use of the legs in standing and walking. The patient cannot get the sole and heel on the ground so as to afford a uniform base of support, and keep his body upright so as to balance it. But this contracture invariably yields to persistent efforts to walk. The palsy of the arms lasts longer than that of the legs, or *vice versâ*, according as the one or other suffers most. The excess of impairment of the muscles most affected is often very conspicuous during recovery. Improvement goes on for a long time, generally until recovery is perfect, but very rarely there is some permanent loss of power. It is remarkable, however, how almost complete a recovery may take place, even after the paralysis has been considerable for a year. The wasting lessens and power returns; latest where there is most loss of irritability, and usually later in the small muscles of the hand than in the muscles of the forearm. Relapses are apt to occur, but only when the cause of the neuritis is still in operation in some degree, or when the patient is again exposed to it or to some adjuvant cause, such as cold.

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[*] Acute gout, it must be remembered, sometimes attacks the larger joints with or before the smaller ones, and may closely resemble an attack of rheumatic fever.

[†] As in a case recorded by Taylor, 'Guy's Hosp. Rep.,' 1888.

[‡] The changes are described by Edmunds and Lawford, 'Trans. Oph. Soc.,' ix, 137.

But the malady sometimes runs a very acute course, especially in the cases due to toxæmia or to cold, so that all parts of the limbs have become weak and the extremities powerless by the end of seven or ten days, and the involvement of the nerves of the respiratory muscles may bring life to an end within a fortnight, sometimes even within a week. In this form there may be severe initial pain, not specially related to the regions afterwards affected. In one case it was severe in the back, in another across the abdomen. It is probably a direct effect of the blood-state, distinct from its influence on the peripheral nerves. Such cases bear considerable resemblance to those of 'acute ascending paralysis" in their course—differing, however, in the fact that the ascension is up the limbs rather than up the general frame. Sometimes, on the other hand, the disease presents a course far more chronic than that described above, and occupies many months in its development and progress. This is often the case with the limited alcoholic forms, especially the ataxic variety, "pseudo-tabes," and with cases in which the toxic agent is absorbed from without very slowly and its effect accumulates gradually, as in some cases of chronic arsenical poisoning. Slight cases, again, treated promptly have been known to recover in a month.

The temperature, usually raised in an acute onset (see p. 120), may be normal in chronic cases, with or without an occasional rise, or may be slightly raised during the chief part of the course of the disease. A persistent considerable elevation suggests some complication, such as phthisis, and should lead to careful and repeated examination of the lungs. It may also indicate a persistence of toxæmia, other indications of which are mentioned among the complications of the disease.

The Cause of Death in acute cases is often palsy of the respiratory muscles, sometimes aided by a catarrhal bronchitis, or paralysis of the heart when the vagus is affected; in more chronic cases it may be caused by exhaustion, due to chronic gastric derangement and the wearying effects of prolonged suffering, or the effects of disease of the spinal cord. Pneumonia frequently causes death in the acute forms, and in alcoholic subjects the inflammation of the lung so often runs a low course, with extensive consolidation, sometimes bilateral and unusual in seat (as in the middle lobe and central portions of the lung), that it is difficult to avoid the suspicion that its form and course are determined by coincident neuritis of the pulmonary branches of the vagus, either primary or secondary to the inflammation of the lung. Death may result unexpectedly from cardiac failure when no preceding symptoms have suggested an affection of the cardiac nerves, and in such cases there is generally a coincident degeneration of the walls of the heart. Fatal neuritis of the vagus, however, may occur very early in relapses. Other coincident effects of chronic alcoholism are frequent causes of a fatal issue, such as cirrhosis of the liver, gastric troubles, and chronic cerebral meningitis in cases of alcoholism, or acetonæmia in cases of diabetic polyneuritis. Various morbid blood-states, causing the neuritis, may continue and cause death. Lastly, phthisis is a frequent cause of death, and probably varies in the relation it bears to the neuritis, sometimes being its consequence, and sometimes apparently a coincident effect of some obscure cause.

COMPLICATIONS.—The chief of these have been just enumerated in the list of the causes of death. The most important in alcoholic cases are the effects of alcohol on various organs and tissues, especially on the liver, heart, and spinal cord. It is very common to find the liver enlarged, sometimes very large; less commonly it is contracted. The enlarged liver may be either fatty or fibroid. Ascites and other effects of portal obstruction are much less frequent complications than might be expected, perhaps because it is rare to have much contraction of the new tissue in the liver. The kidneys also are often diseased. Gastric catarrh with its varied symptoms, morning vomiting, anorexia, and the like, is very common. So also are the several effects of gout due to the alcoholism or to metallic poisons when these have been the cause of the neuritis (as lead, or, as in one case under my notice, silver). On the part of the nervous system there may be various subacute and chronic forms of inflammation of the substance of the spinal cord or of its membranes, the common manifestations of which are to a large extent prevented by the affection of the nerves. The most important, the trunk girdle pains and the affection of the sphincters, are further considered in the section on diagnosis. Chronic cerebral meningitis is also common, and usually fails to produce its usual manifestation, pain, even when marked and extensive opacity and increase of fluid are found after death. It usually causes some chronic mental disturbance, often slight optic neuritis, and sometimes general convulsions. But mental change is also common as a direct effect of the alcohol on the brain; distinct delirium tremens often coincides with the onset of the neuritis. In most alcoholic cases, indeed, the mental state is unnatural; the patient is irritable, intolerant of pain, and deficient in self-control. Various manifestations of hysteria occur in women, and a peculiar childish jocularity is sufficiently common to deserve special mention. The craving for withheld stimulants increases the irritability, and, in the case of women, previous training in duplicity in order to obtain alcohol has usually induced a general deterioration of the moral sense, which has results that startle those who have been successfully deceived. Mental change is for the most part absent in the cases due to cold (save in some in which there is an intense blood-change), and in those produced by metallic poisoning; but it occurs frequently in toxæmic cases, generally in the form of simple delirium, and is apparently due to a simultaneous action of the morbid agent in the cells of the brain. A persistent morbid blood-state may cause endocarditis and other consequences.

Phthisis is also a very frequent complication; its double relation to the neuritis has been already mentioned. The form of lung disease met with in neuritis presents no special peculiarities, except that low pneumonic changes play a considerable part in the morbid process. Acute pneumonia is also common.

PATHOLOGICAL ANATOMY.—The changes in the nerves correspond to those already described in the account of neuritis, with the exception that the chief changes of isolated neuritis are in the connective tissue, and especially in the sheath; and those in the nerve-fibres are subordinate. In most cases of polyneuritis the connective tissue suffers far less than do the nerve-fibres. Considerable variations are found, however, in the extent to which the two elements suffer. In rare cases, and chiefly in those in which the distribution of the affection is not strictly symmetrical, the inflammation chiefly affects the sheath and interstitial tissue; in acute symmetrical cases both the connective tissue and nerve-fibres are involved, as in Fig. 59, p. 132. This was also the condition in the case of fatal septicæmic neuritis recorded by Roth (see p. 115). In the vast majority the fibres are affected chiefly or even exclusively; the changes are essentially parenchymatous. This is the rule when the affection is strictly symmetrical. It is remarkable, often, how slight is the affection of the connective tissue and sheath, even in nerve-trunks that have been extremely tender. Hence it is probable that this tenderness is often due to changes in the nerves of the sheath, the "nervi nervorum," similar to that which the proper conducting fibres of the nerve undergo, rather than to an inflammation of the connective tissue itself.

Naked-eye changes are present only when the connective tissue and sheath are inflamed in considerable degree. In recent and acute cases of this character the nerves may be found reddened, swollen, and sometimes small hæmorrhages are visible. In older cases they may or may not be swollen, but are usually softened, and even pulpy. When the changes are confined to the nerve-fibres, diminished consistence may be the only alteration that can be recognised on simple examination; and often this is absent, and the nerves appear normal on external examination, even when their fibres are extensively diseased. Sometimes under a low magnifying power a section of the nerve has a mottled aspect, due to the irregular degeneration of the fibres.

When the connective tissue is involved the microscope shows the sheath to be infiltrated with lymphoid cells (probably leucocytes), and in old cases many spindle-cells and fibres increase its bulk. The same changes may be traced in the septa between the fasciculi, and in the secondary sheaths that surround the latter. The walls of the vessels are also thickened (Fig. 59, A). When mere acuteness of parenchymatous neuritis causes the connective tissue to be inflamed, the interstitial tissue and inner part of the sheath are chiefly affected, and an amorphous "exudation" may be found in these situations. But the fibres themselves always suffer in conspicuous degree; and in the majority, as in the case from which Figs. 59 and 60 are drawn, they suffer chiefly, the interstitial tissue presenting comparatively little change. In this case the patient was a woman aged thirty-three, alcoholic; the course of the disease was subacute; the symptoms reached a considerable degree in a few weeks, and the patient died, chiefly from coincident liver disease, five months after the onset. The symptoms were characteristic, and continued up to the time of death. The condition found after death is a good illustration of the parenchymatous or degenerative form, but in all forms of polyneuritis the process in the nerve-fibres is the same, whether the connective tissue is or is not affected. The fibres undergo acute degeneration, essentially the same as that which occurs after an injury to a nerve. It has been represented in Figs. 33 and 34 (p. 50), and, associated with inflammation of the sheath, in Fig. 44 (p. 65). Examination in the recent state reveals abundant products of the degeneration of the fibres, granule corpuscles, &c. After hardening, a transverse section, stained with carmine and cleared in the usual way, presents the appearance shown in Fig. 59, A. There is a slight increase in the connective tissue between the fasciculi; the area of these is occupied by tracts of interstitial tissue, which under a higher magnifying power (B) have a branching form, enclosing spaces which have been occupied by nerve-fibres, and in some of which healthy fibres still

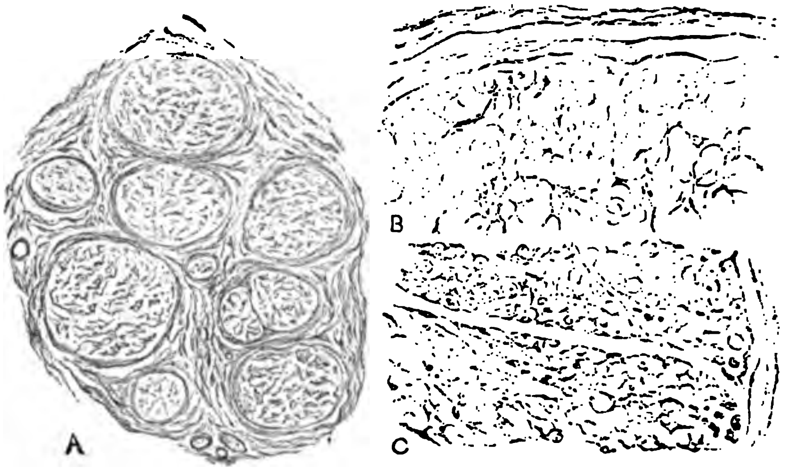
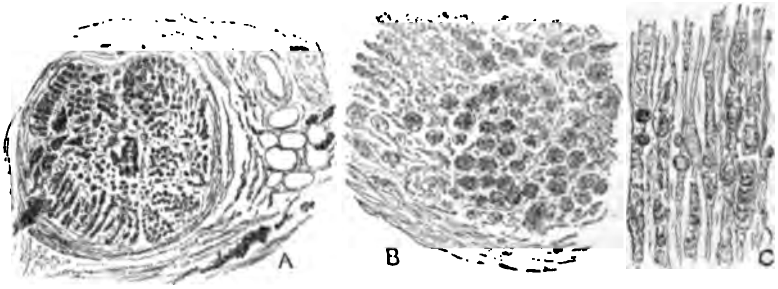


FIG. 59.—Multiple alcoholic neuritis: sections stained with carmine and cleared. A. Transverse section of part of sciatic nerve; low power. B. Part of a fasciculus of same, more highly magnified. C. Part of a less affected fasciculus from a musculo-spiral nerve.



G. 60.—Sections from the same sciatic nerve, stained with osmic acid and mounted in glycerine. A. Fasciculus under low power. B. Part of a fasciculus more highly magnified. C. Longitudinal section of nerve-fibres. (From sections prepared by Dr. Beevor.)

remain (C). The space between this tissue is occupied by the products of degeneration of the fibres, but these are rendered invisible by the clearing process, and only faint indications of their outline can be seen (B). In sections stained with osmic acid and mounted in glycerine (Fig. 60) these products of degeneration, stained dark, are conspicuous. Under a low magnifying power (A) a fasciculus is seen to be studded with black granular spots, elongated where the fibres are divided obliquely; the increase in the tissue between these degenerated fibres is distinct; aggregations of myelin are seen in the connective tissue between the fasciculi. Under higher powers (B) the dark spots are seen to be the sections of degenerated nerve-fibres, in some places two or more being blended into a larger mass. In a longitudinal section (C) the process of degeneration of the fibres is more distinctly seen, and from it the appearance of the transverse section can be better understood. The white substance is broken up and enlarges the fibres at some places, while at others it has been removed, and the sheath is left empty. Round masses of myelin lie (on the left) outside the fibres. The nuclei of the fibres are enlarged; one, of considerable size, is seen in the centre of the figure. Connective-tissue fibres and cells lie between the nerve-clements, and near the sheath are round and spindle cells containing myelin granules and globules which they have taken up (B).

The distribution of the changes in the nerves varies much in different cases. The signs of inflammation of the sheath may exist only on the larger and medium-sized nerves of the limbs, and in the smaller nerves the changes may be confined to the nerve-fibres. These may be traced down into the intra-muscular nerves and their endings. On the larger nerves the signs of inflammation of the sheath are most intense at certain spots, especially (as in simple neuritis) where a nerve turns round a bone, or passes through a fascia, or divides, or in the neighbourhood of joints, their situation being probably determined by mechanical influences.

In all cases in which the affection is chiefly of the nerve-fibres themselves the changes are usually intense in the peripheral parts of the nerves, and as the nerves are examined farther from the periphery the alterations become progressively slighter, and the proportion of normal fibres larger, until the morbid appearance ceases. The centripetal extent of the disease—that is, the degree in which the larger trunks are affected—varies according to the duration and severity of the case. If the nerve-fibres could be isolated and seen like the branches and twigs of a tree, as they separate in the peripheral ramifications, we should see the endings and twig-like branches withered, and as the branchlets become larger and larger by junction they would assume a more and more normal aspect until the larger branches appear perfectly natural. The size of branch to which the change extended would vary according to the severity of the case. In some instances, even those of largest size would present some alteration, while in others the small twigs would be withered in a considerable part of the tree although the change did not extend beyond them. In a tree the leaves would drop off equally whether the twigs only were diseased or the branches also; and so the muscles suffer equally whether the nerve degeneration is limited to the periphery or extends to the nerve-trunks. In the case from which the illustrations are taken even the sciatic nerve presented few healthy fibres; but here, as usual, the anterior roots were healthy. This limitation to the peripheral extremities of the nerves is met with in slight cases of considerable duration, but also in some severe cases of very brief duration. For instance, in a case accompanied by pneumonia, and fatal in seven days, the only change found was in the fibres themselves within the muscles; it was a motor parenchymatous neuritis. The character of the changes is also influenced by that of the nerve-fibres involved. If the affection is partial, as in the ataxic or the purely motor forms, the mixed nerves contain many healthy fibres mingled with those that have undergone atrophy. When the symptoms are both motor and sensory almost all the nerve-fibres are found to have suffered.

The nerves of the limbs alone present changes in the majority of cases. The corresponding nerves on the two sides are almost always affected. The musculo-spiral nerve in the arm and the anterior tibial nerve in the leg usually suffer first and in greatest degree, but often all the terminal nerves of the limbs are involved in some measure; in the lower leg the anterior tibial almost always presents more change than the other nerves of the same level, and in the thigh the sciatic more than the anterior crural. In the arm, the median and musculo-spiral and ulnar all suffer, but the alteration in these nerves in the upper arm may be slight when it is considerable in their branches in the forearm. The changes occasionally met with in other nerves correspond to their implication, as described in the section on symptoms.

The *Muscles* present changes of the same character as those which result from ordinary neuritis (see p. 43). They are paler than normal and smaller in bulk. The fibres are reduced in size, and pale; the transverse striation may be preserved, or they may be granular; normal fibres may be found side by side with those that are degenerated. The nuclei of their sheaths and of the interstitial tissue may be increased in number, and often are arranged in groups; and sometimes a quantity of such nuclei and leucocyte-like cells may separate widely the fibres, and granular and pigment masses may accumulate between them. In very acute cases the changes in the muscles may be great, and alike parenchymatous and interstitial; the fibres presenting loss of their striation with fatty and granular degeneration, while there may be a great increase in the amount of the interstitial tissue, especially of its nuclear elements.

The *Spinal Cord* has been found healthy in most cases. Occasionally the grey substance has appeared pale and somewhat shrunken, so as to present a slighter difference from the white than normal. The ganglion-cells, in some instances, have presented abnormal appearances—a vitreous appearance or vacuolation, and this when the anterior nerve-roots were not diseased. In many alcoholic cases, however, changes more or less considerable have been found in the cord. Chronic myelitis in irregular disseminated areas is the chief, and there has been sometimes also chronic inflammation of the membranes, especially of the pia mater, involving also the peripheral layers of the cord; an instance of this is figured in the chapter on chronic myelitis. In other cases there has been merely a general increase in the connective tissue throughout the cord, which has been in some instances greater in one region than elsewhere, and especially marked in the posterior columns.

Other organs are often found diseased, and in alcoholism the lesions common in that disease are generally found in some degree, especially alterations in the liver and kidneys. The former may be large and either cirrhosed or fatty; the kidneys are generally large and opaque, sometimes with fatty striæ in the cortex. Pneumonia is frequent both in these and in all cases due to causes that induce an intense change in the blood. In these there may also be enlargement of the spleen, such as is met with in typhoid fever and septicæmia. Phthisical changes in the lungs are also common in chronic cases, both in those that are due to alcohol and in others. No special peculiarity has been observed in the pulmonary lesion, but it is very desirable that more attention should be paid to the state of the nerves of the lungs entering the diseased parts. When not otherwise diseased, the lungs are generally found the seat of terminal congestion and œdema. The walls of the heart may be degenerated, and alterations may sometimes be found in its nerves.

Pathology.—An outline of the chief facts in pathology of multiple neuritis has been given in the introductory section, and not much remains to be added. The relation of the motor symptoms to the lesion of the nerves has also been described in the general account of diseases of the nerves, and in the description of isolated neuritis; all that has been said of the single form is true also of that which is multiple. The wasting that accompanies the loss of power, and the change in electrical irritability, are the same in each. In each it is true, moreover, that the position of the lesion in the course of the nerve-fibre makes no difference to the symptoms; these are the same whether the disease is in the trunk of the nerve, the peripheral fibres, or the actual nerve-endings. The relation of the ataxy in the pseudo-tabetic form to a neuritis of the afferent muscular nerves is proved partly by exclusion, partly by analogy, since in true tabes the lesion may be confined to these; the spinal cord may be free, and only the peripheral nerves diseased. The evidence for this will be described in the general account of the functions and symptoms of diseases of the spinal cord, and in the chapter on locomotor ataxy—a disease which sometimes not only resembles, but is almost identical with the pseudo-tabetic form of neuritis. The pains and hyperæsthesia are explained by the fact that the disease of the nerves is one in which the nerve-fibres themselves suffer primarily, and that on them the cause must have a primary and direct action.* An influence which leads to the molecular destruction of the nerve-fibres must, in destroying their structure, disturb gravely their function; and this disturbance of structure and of function proceeds in the parts still connected with the nerve-centres, so that the irritative influence of the molecular changes can act on the centres by the normal upper parts of the nerves throughout the whole course of the malady. It induces in the centres, as all prolonged pain does, an undue readiness of action, a state of over-excitability; and so we can understand the persistence of the pains and hyperæsthesia, and the fact that these continue until the process of restoration has become definitely established. The same considerations apply to the tenderness of the muscles, since the afferent muscular nerves correspond in the direction of their function to the sensory nerves of the skin. In both cases mechanical influences constitute the chief mode of their normal stimulation, and hence they may well be morbidly sensitive to such influences. The peripheral distribution of the changes indicates, moreover, that probably the first to suffer are those in which functional susceptibility is most highly developed—the actual endings of the nerves, and the structures which are especially adapted for the reception of stimuli and the production of nerve-force

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* In the first edition I suggested that the degeneration of some of the fibres might be secondary to their damage by the inflammation of the sheath and interstitial tissue at some higher spot. But this now seems very unlikely; it is far

We have seen that two facts give us the key to the pathology of the disease so far as this is yet understood. One is its symmetry, the other is the fact that it is commonly a disease of nerve-fibres—that these, and not the connective tissue, are the parts primarily and specially diseased. In all structures, in proportion to the intensity and acuteness of the parenchymatous affection, the interstitial tissue participates in the changes, and escapes in proportion to its slowness and slightness. The extent to which this is true of multiple neuritis has been already pointed out. Further, in its relation to a morbid blood-state as its sole cause, we have the key to the symmetry of the affection as well as to its parenchymatous nature, and on this point there remains little to add to what has been already said. Such a limitation to the nerve-fibres as is seen in multiple neuritis is unknown in the isolated form, in which, as in gouty or syphilitic neuritis, the affection is chiefly one of the sheath and interstitial connective tissue; and the fibres suffer only secondarily in chronic cases, although, in accordance with the law already stated, they are involved in acute cases in proportion to the intensity of the process.

It is not surprising that the symptoms of multiple neuritis should bear so close a resemblance to those of some affections of the spinal cord, since we must regard the peripheral nerves, at least the motor fibres, as essentially outlying parts of the cord, and it will be seen that one central disease, primary lateral sclerosis, presents many points of correspondence with the chronic form of polyneuritis, being

more probable that all the fibres suffer in the same way, and the more thoroughly cases are studied the more clearly does it appear that all symmetrical neuritis is peripheral and parenchymatous.

nerves and [illegible]
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of the blood-state may have a considerable influence in ts effects upon the nerves and ameliorating the course of the

nosis of multiple neuritis depends on the motor and sensory above described, their correspondence in function and dis- in the opposite limbs, their peculiar symmetrical localisation remities in the first instance, and on the tenderness of the trunks, and muscles. Symmetrical weakness of the extensor situated in the forearm, and of the corresponding muscles in r leg, or of either of these, is the leading diagnostic motor sym- lateral wrist-drop or foot-drop suggests multiple neuritis, and hesia or deep tenderness gives strength to the opinion. It is, most important to remember how widely the symptoms vary different forms and in different cases of the same form. The lly fatal cases, with palsy quickly becoming general, are of a unlike the chronic cases in which one group of muscles on ffers alone; while there is a positive contrast between the a case of "pseudo-tabes," in which ataxy and pains are almost mptoms. In chronic cases also the early pains are often or those of rheumatism, and in those of acute course and due hese pains suggest the onset of rheumatic fever rather than ffection of the nerves. The seat of the pains, however, is not rheumatic pain, and their association with tingling in the ities should excite a suspicion that they are of nerve origin efore muscular weakness renders their nature clear. The im- ance is great of ascertaining precisely the position of all pains in mbs. Neglect to do so is a fertile source of error in diagnosis, may easily put the observer on a wrong path. from which other ptoms fail to withdraw him. It must be again pointed out that n near a joint, produced by movement of the joint, is easily mis- en for pain in the joint, even when it depends entirely on tenderness the adjacent nerves. In some cases the pain is regarded as uralgic, especially when it is seated in the arms and hands, and ts position is perceived to be that of nerve-trunks or branches. But the general elements of the diagnosis between the two diseases generally (described partly in the section on neuritis, partly in that on neuralgia in vol. ii) usually suffice for the distinction, the most important point being the persistence of tenderness and hyperæsthesia in relative excess of the spontaneous pain, and the development of symptoms that indicate damage to the conducting fibres. The bilateral symmetry of the pain should also help to prevent error; true neuralgia is essentially a unilateral disease. A gouty diathesis may increase the difficulty of the diagnosis, especially in alcoholic cases; but the pains of gout are unequivocally articular or muscular, and neuritis due to it is single and distinctly adventitial.

The symptoms resemble most closely those of certain diseases of the spinal cord, especially acute and subacute inflammation of the grey [illegible] nearly, indeed, all cases of multiple [illegible] that nature. It is chiefly in the rheu- [illegible] that the diagnosis is really difficult. In each [illegible] muscular wasting with the reac- [illegible] rheumatic pains, and a tendency to the [illegible] affected parts. The distinction rests [illegible] localisation of the neuritic palsy, while that of [illegible] random in distribution; on the [illegible] neuritic pains; on the tenderness of [illegible] and on the changes in sensibility, a symptom [illegible] The electrical reactions are the same [illegible] seen, and the early extinction of voltaic [illegible] (see p. 124) is met with only in disease of [illegible] incorrect; I have more than once met with it [illegible] But we have seen that it is probable that [illegible] sometimes inflamed in polio-myelitis, and suffer [illegible] the cause of the spinal lesion. In this con- [illegible] spinal symptoms preponderate, and the distri- [illegible] irregular. In all cases the presence of [illegible] irritability, or of the spasm in the legs that accom- [illegible] degrees of this, may be taken as proof of disease of [illegible] muscular spasm, indeed, has this significance. [illegible] paralysed, even though the paralysis closely [illegible] and the legs are weak, with excessive [illegible] the disease is of the cord, and not of the [illegible] presented by some sensory cases [illegible] tingling and numbness in the legs, and [illegible] cord disease in the presence of [illegible] The latter will be considered further on. The [illegible] the presence or absence of the knee- [illegible] In peripheral neuritis the sensory symptoms [illegible] symmetrical than in cord disease; there is [illegible] anaesthesia, for instance, in the palms or soles, or both, [illegible] of the nerves and muscles will generally [illegible] characteristic.

[illegible] the nerve-roots, may cause paralysis, [illegible] all four limbs are not affected; [illegible] the anaesthesia extends on to the upper part [illegible] the trunk. There is no tenderness of nerve- [illegible] distinct evidence of damage to the spinal [illegible] disease, "acute ascending paralysis," [illegible] multiple neuritis, but in the [illegible] from the legs to the arms, and is not [illegible] at the same time or successively, and [illegible] the usual form of multiple neuritis. [illegible] in the spinal [illegible] in degree, may also be attended by muscular

wasting and anæsthesia, but there is no considerable pain, and the weakness in the limbs usually succeeds paralysis of the palate and ciliary muscle, never met with in other forms of polyneuritis. The distinction from other diseases (as, for instance, that from the symmetrical paralysis of the extensors which may occur at the onset of progressive muscular atrophy) will be considered when these diseases are described.

The distinction of the ataxic form, "pseudo-tabes," from true tabes is often beset with great difficulty. This is indeed natural, since the lesion in true tabes may be identical with that in the neuritic affection; not only is degeneration of the peripheral nerves a common lesion in tabes, having the same distribution at the peripheral extremities of the nerves as in polyneuritis, but in the variety termed "neuro-tabes" the lesion consists only in this nerve-degeneration, and the spinal cord is free from disease. We have then affections identical in pathological anatomy, and almost identical in symptoms. Yet we must class the neuro-tabes with the spinal form, from which polyneuritis, as here described, has to be distinguished; it is necessary to do so on account of the relations and etiology of the two maladies, as well as on account of practical considerations. The presence of actual paralysis, of distinct weakness of the extensors, in addition to inco-ordination, would of course decide the question, since there is no actual loss of power in tabes: hence a distinction in gait pointed out by Westphal and Charcot, that in pseudo-tabes the feet are raised too high on account of the difficulty of getting the toes off the ground (like a person stepping over low objects), is of little actual value; it only exists when there is distinct loss of power. But it is in the cases of neuritis in which there are only sensory symptoms that the difficulty especially exists. It occurs, moreover, chiefly when the ataxy is moderate in degree; when this is so great that the patient is not able to stand alone, in spite of the power of moving all joints of the legs with fair force, true tabes should alone be thought of. The cases of tabes in which there is moderate inco-ordination of movement, distinct unsteadiness on walking, whether increased by closure of the eyes or not, and distinct irregularity and uncertainty in the movement of the feet and legs, are those which are perfectly simulated by the pseudo-tabetic form of polyneuritis. Pains are common to the two diseases, and neither prolonged acute pain nor dull rheumatoid pains afford the means of distinction. True "lightning" pains are seldom met with in neuritis; they are therefore strong evidence that the case is one of tabes. Extreme hyperæsthesia is much more common in neuritis than in tabes, but is of little service in the diagnosis because it is not often present in ataxic polyneuritis, but the muscular tenderness of neuritis is of considerable diagnostic value. A great excess of the reflex action from the skin is occasionally met with in tabes, the sensory impressions being painless; while in ataxic neuritis the reflex

action is seldom excessive, and is generally diminished, while the sensory impression is commonly painful from the hyperæsthesia. Some other symptoms are also of value as indications of tabes rather than neuritis, —an affection of the sphincters; a sense of constriction round the trunk (which may be felt over a wide area as a band rather than as a cord); and lastly (but among the first in importance), the reflex action of the pupil to light is often lost; scarcely ever in neuritis. Since it may be normal in tabes, its loss is of more significance than its presence, but the cases of tabes in which it is preserved form only a small minority, so that its preservation is often at least corroborative evidence that the case is one of polyneuritis. Mental change is of little value in the diagnosis, unless it is in the form of definite delirium; but the less common symptoms of tabes, if present, often afford proof of the nature of the malady, although generally in association with other symptoms rather than by themselves. Optic nerve atrophy, for instance, is almost unknown in the neuritic "pseudo-tabes," and so also are the various "crises:" care must be taken not to confound the vomiting of alcoholic gastric disturbance with the gastric crises of tabes. The trophic changes differ in the two, the tabetic enlargement of the bones and disease of the joints are unknown in neuritis; and so, in the former affection, are the simple arthritic adhesions and the "glossy skin" of the latter. If all the symptoms are considered separately for their individual value, and conjointly for their associated significance, it is not often that the observer will remain in doubt.

One other special diagnosis should be mentioned, that for hysterical palsy. This occurs chiefly in women when the symptoms are motor and are in the legs. When in the arms, the characteristic situation of the palsy usually attracts attention and prevents error. A defect in the power of extension of the wrist has probably never been met with as a consequence of hysteria. Slight ataxy may, however, be purely functional in nature, but the knee-jerk is not lost, and if there is anæsthesia it is much more extensive in the functional affection.

It is of great importance to know whether the spinal cord is suffering as well as the nerves. The following symptoms are those that are the most common and important evidence of this complication:— (1) Any impairment of the functions of the bladder or rectum—incontinence of fæces, retention or incontinence of urine. It is not, indeed, improbable that their impairment may form part of the most intense form of polyneuritis; but it is not part of the ordinary phenomena of the disease; hence, in such cases, these symptoms suggest that the disease involves the cord as well as the nerves. It is important to remember that when there is mental change, evacuations into the bed are frequently without significance as to the state of the sphincters, and are due solely to the inertia of the mind. This is a fertile source of error. (2) A distinct sense of constriction, "girdle-pain," referred to any part of the trunk, probably always constitutes proof of damage to the cord. Of the same significance is sharp pain radiating around the trunk at a definite level and not elsewhere. (3) An important distinction in alcoholic cases is an unusual distribution of the affection, *e. g.* complete paralysis of all parts of the legs, or weakness of the muscles above the knee, and not of those below. Impairment of sensation up to a certain level on the legs or trunk has the same significance; disease of the nerves does not involve those of sensation up to a definite level. Bedsores also increase the probability that the cord is affected.

If a case is recognised as one of multiple neuritis there remains the question, what is its cause? In many cases this question does not arise, because the cause is obtrusive, and has been recognised as one of the elements of the diagnosis; but in other cases the cause is not clear, the diagnosis has been made independently of it, and it remains to be discovered. For the most part it is only necessary to know the various causes in order to determine which is effective in the patient under observation, because one or another of them is to be discovered without difficulty, and the rest are absent. It may be useful, however, to remember that, as a rule, in chronic metallic poisoning the arms suffer before the legs, and that in lead poisoning the acute paralysis is limited to the arms; in arsenical poisoning the affection of the arms is soon followed by that of the legs, while in alcoholic cases the legs generally suffer first. In a case in which arms and legs are affected simultaneously metallic poisoning would be a very unlikely cause. An initial affection of the proximal parts of the limbs at the same time as the distal parts suggests some other cause than alcohol, especially cold or toxæmia. Early affection of the muscles of the trunk or of the bulbar nerves has the same significance, and so also has severe constitutional disturbance at the outset. It must be also remembered, however, that alcoholism often co-operates with other causes, especially with cold; and it must also be remembered that the inquiry needs to be exhaustive before the influence of alcohol can be excluded. This is especially true in the case of females, who often obstinately deny that which would bring shame upon them. Exposure to cold has generally been severe; and if it has not, the co-operation of alcohol is extremely probable. Neuralgic pains of irregular distribution in addition to the symptoms of polyneuritis should suggest diabetes as a cause, but the examination of the urine for sugar should never be omitted. When polyneuritis is due to septicæmia this cause may be suggested by the fact that the patient is under treatment for some surgical ailment; while in all cases of obscure origin inquiry should be made for any acute specific disease to which the neuritis is known to be secondary, and the fact should be remembered that a considerable interval may separate the two. Some form of toxæmia is always suggested by the fact that the onset of the neuritis is associated with nent, since multiple neuritis is not known to be excited by any other organic poison than that which is so produced.

The forms of polyneuritis ascribed to anæmia and cachexia, &c., may be a simple failure of nutrition in those structures of the nervous system that are farthest removed from the centres; but the possibility must not be forgotten that imperfect tissue changes may generate a toxic agent capable of acting on these nerves, analogous to that assumed to be effective in diabetes.

The changes in nutrition are sufficiently explained by the alterations found in the nerves, in accordance with the general facts of trophic disturbance stated at pp. 22 and 61. That in the muscles corresponds in distribution to the changes in the motor nerves, that in the skin and joints to those in the sensory nerves. All trophic changes which result from ordinary nerve-lesions must be ascribed to the propagation downwards of the influence of the defective and abnormal nutrition; and hence it necessarily follows that such abnormal nutrition, beginning in the nerve-endings, should have a similar influence, since it is through those nerve-endings that the effects of higher lesions are transferred to the tissues. The law already stated holds good here also,—that changes in nutrition are in proportion to the intensity and acuteness of the disturbance in the nerves. All that has been said regarding such changes in the skin, in diseases of isolated nerves, is true also of the multiple form, and even more clearly in the case of the motor nerves and muscles. The more acute the process in the nerves, the more intense is the change in the muscular tissue. One special point, indeed, arises in this connection. Although, in very acute isolated neuritis, the muscles may undergo the same intense and destructive changes seen in the most acute forms of multiple neuritis, it is possible that the extreme changes in the latter are sometimes a result and evidence of a direct action of the blood-poison on the muscular tissue itself.* There are some facts of both physiology and pathology which suggest what may be called a certain degree of solidarity of relation between the substance of the nerve-endings and that of the muscular fibres on which they terminate. This may lead to a common susceptibility to suffer from the same morbid influence, so far less in the muscle that only when the influence is very intense does its nutrition suffer directly to a considerable extent.

Diagnosis.—There are few diseases in which an early diagnosis is of greater importance. Prompt treatment would save many patients from months of suffering and disability, since the removal of the cause ensures with certainty, during a gradual onset, a quick removal of the symptoms, whereas its effect on the developed disease is manifested much more slowly. These remarks apply especially to the alcoholic cases, but in some of the others the patient may be saved further exposure to the cause if the disease is promptly recognised, or early treatment of the blood-state may have a considerable influence in lessening its effects upon the nerves and ameliorating the course of the affection.

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* This suggestion has been made by Soemerling as an explanation of the very intense changes found in the muscles in one acute case.

The diagnosis of multiple neuritis depends on the motor and sensory symptoms above described, their correspondence in function and distribution in the opposite limbs, their peculiar symmetrical localisation in the extremities in the first instance, and on the tenderness of the skin, nerve-trunks, and muscles. Symmetrical weakness of the extensor muscles situated in the forearm, and of the corresponding muscles in the lower leg, or of either of these, is the leading diagnostic motor symptom. Bilateral wrist-drop or foot-drop suggests multiple neuritis, and hyperæsthesia or deep tenderness gives strength to the opinion. It is, however, most important to remember how widely the symptoms vary both in different forms and in different cases of the same form. The acute rapidly fatal cases, with palsy quickly becoming general, are of a type quite unlike the chronic cases in which one group of muscles on each side suffers alone; while there is a positive contrast between the latter and a case of "pseudo-tabes," in which ataxy and pains are almost the only symptoms. In chronic cases also the early pains are often mistaken for those of rheumatism, and in those of acute course and due to cold these pains suggest the onset of rheumatic fever rather than of any affection of the nerves. The seat of the pains, however, is not that of rheumatic pain, and their association with tingling in the extremities should excite a suspicion that they are of nerve origin even before muscular weakness renders their nature clear. The importance is great of ascertaining precisely the position of all pains in the limbs. Neglect to do so is a fertile source of error in diagnosis, and may easily put the observer on a wrong path, from which other symptoms fail to withdraw him. It must be again pointed out that pain near a joint, produced by movement of the joint, is easily mistaken for pain in the joint, even when it depends entirely on tenderness of the adjacent nerves. In some cases the pain is regarded as neuralgic, especially when it is seated in the arms and hands, and its position is perceived to be that of nerve-trunks or branches. But the general elements of the diagnosis between the two diseases generally (described partly in the section on neuritis, partly in that on neuralgia in vol. ii) usually suffice for the distinction, the most important point being the persistence of tenderness and hyperæsthesia in relative excess of the spontaneous pain, and the development of symptoms that indicate damage to the conducting fibres. The bilateral symmetry of the pain should also help to prevent error; true neuralgia is essentially a unilateral disease. A gouty diathesis may increase the difficulty of the diagnosis, especially in alcoholic cases; but the pains of gout are unequivocally articular or muscular, and neuritis due to it is single and distinctly adventitial.

The symptoms resemble most closely those of certain diseases of the spinal cord, especially acute and subacute inflammation of the grey matter, polio-myelitis. Until recently, indeed, all cases of multiple neuritis were looked upon as of that nature. It is chiefly in the rheumatic and toxæmic forms that the diagnosis is really difficult. In each disease there may be a febrile onset, muscular wasting with the reaction of degeneration, initial rheumatic pains, and a tendency to the spontaneous recovery of the least affected parts. The distinction rests on the symmetrical localisation of the neuritic palsy, while that of polio-myelitis is characteristically random in distribution; on the persistence and severity of the neuritic pains; on the tenderness of the inflamed nerve-trunks; and on the changes in sensibility, a symptom never present in polio-myelitis. The electrical reactions are the same in both affections. It has been said the early extinction of voltaic irritability in the muscle (see p. 124) is met with only in disease of the nerves, but this is incorrect: I have more than once met with it in central affections. But we have seen that it is probable that the nerve-trunks are sometimes inflamed in polio-myelitis, and suffer as a simultaneous effect of the cause of the spinal lesion. In this condition, however, the spinal symptoms preponderate, and the distribution of the neuritis is irregular. In all cases the presence of increased myotatic irritability, or of the spasm in the legs that accompanies considerable excess of this, may be taken as proof of disease of the cord; all active muscular spasm, indeed, has this significance. Thus if the arms are paralysed, even though the paralysis closely resembles that of polyneuritis, and the legs are weak, with excessive knee-jerk and foot-clonus, the disease is of the cord, and not of the nerves. The greatest difficulty is presented by some sensory cases in which there are only tingling and numbness in the legs, and also by the problem of detecting cord disease in the presence of peripheral neuritis. The latter will be considered further on. The former is generally decided by the presence or absence of the knee-jerk, and by the fact that in peripheral neuritis the sensory symptoms are more peculiar and symmetrical than in cord disease; there is tingling or anæsthesia, for instance, in the palms or soles, or both, and a careful examination of the nerves and muscles will generally reveal a condition that is characteristic.

Pachymeningitis, damaging the nerve-roots, may cause paralysis, wasting, and anæsthesia, but in this all four limbs are not affected; the legs rarely suffer; the anæsthesia extends on to the upper part of the limbs and to the trunk; there is no tenderness of nerve-trunks, and there is usually distinct evidence of damage to the spinal cord itself. The mysterious disease, "acute ascending paralysis," may resemble the most rapid form of multiple neuritis, but in it the symptoms ascend the trunk from the legs to the arms, and do not begin in the hands and feet at the same time or successively, and involve the trunk last, as does the usual form of multiple neuritis. Moreover there is no anæsthesia in the spinal malady. Diphtheritic paralysis, when severe in degree, may also be attended by muscular

wasting and anæsthesia, but there is no considerable pain, and the weakness in the limbs usually succeeds paralysis of the palate and ciliary muscle, never met with in other forms of polyneuritis. The distinction from other diseases (as, for instance, that from the symmetrical paralysis of the extensors which may occur at the onset of progressive muscular atrophy) will be considered when these diseases are described.

The distinction of the ataxic form, "pseudo-tabes," from true tabes is often beset with great difficulty. This is indeed natural, since the lesion in true tabes may be identical with that in the neuritic affection; not only is degeneration of the peripheral nerves a common lesion in tabes, having the same distribution at the peripheral extremities of the nerves as in polyneuritis, but in the variety termed "neuro-tabes" the lesion consists only in this nerve-degeneration, and the spinal cord is free from disease. We have then affections identical in pathological anatomy, and almost identical in symptoms. Yet we must class the neuro-tabes with the spinal form, from which polyneuritis, as here described, has to be distinguished; it is necessary to do so on account of the relations and etiology of the two maladies, as well as on account of practical considerations. The presence of actual paralysis, of distinct weakness of the extensors, in addition to inco-ordination, would of course decide the question, since there is no actual loss of power in tabes: hence a distinction in gait pointed out by Westphal and Charcot, that in pseudo-tabes the feet are raised too high on account of the difficulty of getting the toes off the ground (like a person stepping over low objects), is of little actual value; it only exists when there is distinct loss of power. But it is in the cases of neuritis in which there are only sensory symptoms that the difficulty especially exists. It occurs, moreover, chiefly when the ataxy is moderate in degree; when this is so great that the patient is not able to stand alone, in spite of the power of moving all joints of the legs with fair force, true tabes should alone be thought of. The cases of tabes in which there is moderate inco-ordination of movement, distinct unsteadiness on walking, whether increased by closure of the eyes or not, and distinct irregularity and uncertainty in the movement of the feet and legs, are those which are perfectly simulated by the pseudo-tabetic form of polyneuritis. Pains are common to the two diseases, and neither prolonged acute pain nor dull rheumatoid pains afford the means of distinction. True "lightning" pains are seldom met with in neuritis; they are therefore strong evidence that the case is one of tabes. Extreme hyperæsthesia is much more common in neuritis than in tabes, but is of little service in the diagnosis because it is not often present in ataxic polyneuritis, but the muscular tenderness of neuritis is of considerable diagnostic value. A great excess of the reflex action from the skin is occasionally met with in tabes, the sensory impressions being painless; while in ataxic neuritis the reflex

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action is seldom excessive, and is generally diminished, while the sensory impression is commonly painful from the hyperæsthesia. Some other symptoms are also of value as indications of tabes rather than neuritis, —an affection of the sphincters; a sense of constriction round the trunk (which may be felt over a wide area as a band rather than as a cord); and lastly (but among the first in importance), the reflex action of the pupil to light is often lost; scarcely ever in neuritis. Since it may be normal in tabes, its loss is of more significance than its presence, but the cases of tabes in which it is preserved form only a small minority, so that its preservation is often at least corroborative evidence that the case is one of polyneuritis. Mental change is of little value in the diagnosis, unless it is in the form of definite delirium; but the less common symptoms of tabes, if present, often afford proof of the nature of the malady, although generally in association with other symptoms rather than by themselves. Optic nerve atrophy, for instance, is almost unknown in the neuritic "pseudo-tabes," and so also are the various "crises:" care must be taken not to confound the vomiting of alcoholic gastric disturbance with the gastric crises of tabes. The trophic changes differ in the two, the tabetic enlargement of the bones and disease of the joints are unknown in neuritis; and so, in the former affection, are the simple arthritic adhesions and the "glossy skin" of the latter. If all the symptoms are considered separately for their individual value, and conjointly for their associated significance, it is not often that the observer will remain in doubt.

One other special diagnosis should be mentioned, that for hysterical palsy. This occurs chiefly in women when the symptoms are motor and are in the legs. When in the arms, the characteristic situation of the palsy usually attracts attention and prevents error. A defect in the power of extension of the wrist has probably never been met with as a consequence of hysteria. Slight ataxy may, however, be purely functional in nature, but the knee-jerk is not lost, and if there is anæsthesia it is much more extensive in the functional affection.

It is of great importance to know whether the spinal cord is suffering as well as the nerves. The following symptoms are those that are the most common and important evidence of this complication:—(1) Any impairment of the functions of the bladder or rectum—incontinence of fæces, retention or incontinence of urine. It is not, indeed, improbable that their impairment may form part of the most intense form of polyneuritis; but it is not part of the ordinary phenomena of the disease; hence, in such cases, these symptoms suggest that the disease involves the cord as well as the nerves. It is important to remember that when there is mental change, evacuations into the bed are frequently without significance as to the state of the sphincters, and are due solely to the inertia of the mind. This is a fertile source of error. (2) A distinct sense of constriction, "girdle-pain," referred to any part of the trunk, probably always constitutes proof of damage to the cord. Of the same significance is sharp pain radiating around the trunk at a definite level and not elsewhere. (3) An important distinction in alcoholic cases is an unusual distribution of the affection, *e. g.* complete paralysis of all parts of the legs, or weakness of the muscles above the knee, and not of those below. Impairment of sensation up to a certain level on the legs or trunk has the same significance; disease of the nerves does not involve those of sensation up to a definite level. Bedsores also increase the probability that the cord is affected.

If a case is recognised as one of multiple neuritis there remains the question, what is its cause? In many cases this question does not arise, because the cause is obtrusive, and has been recognised as one of the elements of the diagnosis; but in other cases the cause is not clear, the diagnosis has been made independently of it, and it remains to be discovered. For the most part it is only necessary to know the various causes in order to determine which is effective in the patient under observation, because one or another of them is to be discovered without difficulty, and the rest are absent. It may be useful, however, to remember that, as a rule, in chronic metallic poisoning the arms suffer before the legs, and that in lead poisoning the acute paralysis is limited to the arms; in arsenical poisoning the affection of the arms is soon followed by that of the legs, while in alcoholic cases the legs generally suffer first. In a case in which arms and legs are affected simultaneously metallic poisoning would be a very unlikely cause. An initial affection of the proximal parts of the limbs at the same time as the distal parts suggests some other cause than alcohol, especially cold or toxæmia. Early affection of the muscles of the trunk or of the bulbar nerves has the same significance, and so also has severe constitutional disturbance at the outset. It must be also remembered, however, that alcoholism often co-operates with other causes, especially with cold; and it must also be remembered that the inquiry needs to be exhaustive before the influence of alcohol can be excluded. This is especially true in the case of females, who often obstinately deny that which would bring shame upon them. Exposure to cold has generally been severe; and if it has not, the co-operation of alcohol is extremely probable. Neuralgic pains of irregular distribution in addition to the symptoms of polyneuritis should suggest diabetes as a cause, but the examination of the urine for sugar should never be omitted. When polyneuritis is due to septicæmia this cause may be suggested by the fact that the patient is under treatment for some surgical ailment; while in all cases of obscure origin inquiry should be made for any acute specific disease to which the neuritis is known to be secondary, and the fact should be remembered that a considerable interval may separate the two. Some form of toxæmia is always suggested by the fact that the onset of the neuritis is associated with such pyrexia and constitutional disturbance as indicate an acute morbid blood-state. But this diagnosis often rests chiefly on the exclusion of other causes; if these are absent the fact that neuritis is known to result from obscure blood-poisons may be allowed weight, especially if the symptoms begin in the legs or in all parts of the limbs simultaneously, and do not affect exclusively at first the extensors of the extremities. Sensory symptoms in these cases at the early stage often take the form of tingling and "numbness," and the pains so characteristic of the alcoholic form are frequently absent, or are represented only by dull aching. In slight cases of this type sensations of numbness and "pins and needles" in the legs, with readiness of fatigue and loss of the knee-jerk, may be the only symptoms. The facts that the nature of toxæmic causes is to a large extent unknown, and that the poison may probably be acquired from without, make it necessary to be prepared to find this cause of multiple neuritis under almost any circumstances. Residence abroad should suggest an inquiry for exposure to malarial influences. The possible significance of tubercle as a cause should lead to a careful examination of the lungs; but the possibility that phthisis may be an effect, and not a cause, of multiple neuritis, must be borne in mind.

PROGNOSIS.—The danger to life is in proportion to the acuteness of the malady, to its severity as measured by the extent of the paralysis, and to the degree in which the strength of the patient is impaired by other maladies, or by the cause of the neuritis. It is important also to remember that when the affection has increased up to the time that the patient comes under treatment, it generally does not respond immediately to the arrest of its cause, such as the withdrawal of alcohol, but continues to increase for two or three weeks. It is this temporary progressive tendency that constitutes the graver element in the disease, and must be taken into account in every initial prognosis. Hence the forecast must be guarded in all cases that are severe when the patient comes under treatment, and most so if there has been a rapid increase in symptoms that had existed for a long time in slight degree. If the extensors of hands and feet are powerless, and the muscles connecting the limb with the trunk are distinctly enfeebled, unless the disease has clearly begun to lessen, there is danger that a further extension may involve the muscles of respiration; this is especially great when the muscles moving the shoulder are considerably weakened. It obviously follows that the danger is still greater if the muscles of the trunk, and especially of the chest, have already given evidence of changes in their nerves. Pain in the trunk of the same character as that in the limbs is also a grave symptom if the motor power of the limbs has become small, or the case is one unusually general in its distribution, because where the sensory fibres are suffering the motor fibres are in danger also. Paralysis of the diaphragm adds considerably to the danger, and its power should be carefully watched in the manner and with the precautions mentioned in the account of its paralysis (p. 27). It may become paralysed without the fact coming to the consciousness of a patient lying quietly in bed, but a slight weakness of the intercostals will then cause considerable difficulty of breathing and an accumulation of mucus in the lungs, a condition predisposing to definite bronchitis, by which the patient may be quickly suffocated. The importance of this point is great, since respiratory palsy is one of the most common immediate causes of death. It is probable also that deficient breathing power is one of the causes, perhaps even the chief cause, that leads to the development of the chronic phthisical lung disease that comes on in so many prolonged cases. Especially important, also, is any indication that the nerves of the heart are suffering. In an acute increase due to continued use of alcohol, the vagus sometimes suffers severely before other nerves, and increasing frequency of the pulse is then of most serious significance. The prognosis should be guided by the motor rather than by the sensory symptoms. The latter may improve while the former increase, without lessening the gravity of the latter indication. Persistent œdema is of bad omen, and so also are other indications of considerable blood-change.

When once the malady has become stationary, and its cause has definitely ceased, the danger to life is small, except in severe cases, in which the pains are intense, the patient helpless, or the heart is feeble, and the pulse persistently frequent, or there are signs of lung disease. In these, as in all cases, moreover, the danger is much increased by any indication that the spinal cord is involved. The tendency to recovery is far less in the cord than in the nerves; and retention of urine (with all its secondary consequences), bedsores, &c., are prone to occur, and to involve their own danger even if the disease in the cord does not increase. The prognosis as regards life in cases of moderate severity depends, indeed, as much on complications as on the state of the nerves. Signs of chronic cerebral meningitis, of degeneration of the walls of the heart, of disease of the liver and of the kidneys, also lessen the prospect of escape with life.

The danger is, however, greatest, and the mortality highest in the acute cases that are due to some toxæmic state or to cold, and it must be estimated by the rapidity with which the paralysis develops and spreads. Whenever it becomes considerable in a few days, the danger of early extension to the mechanism for respiration is very great. In these cases, also, pneumonia frequently occurs, concomitant with the early symptoms, and few cases thus complicated recover.

In cases that are not fatal, the full development of the symptoms is followed by a stationary period which lasts for one or two months before improvement begins, and for this the patient or friends should be prepared. The course of restoration is always extremely slow; the muscles least affected regain power in from two to four months, but it is usually six months or more from the onset of improvement before the extensors are fairly strong, if their palsy was considerable or complete. The intrinsic muscles of the hand are still longer in recovery. The improvement in the nutrition of the muscles follows that in their power, so that they remain smaller than normal, long after they have regained strength. It may indeed be years before the muscles whose size can be well seen, such as the abductor indicis, are of normal size. It is seldom that any permanent weakness is left unless the spinal cord is diseased, and when a patient is distinctly improving a good ultimate prognosis may be given, provided there are no complications to interfere with it. In no case with distinct spinal symptoms can we feel confident that restoration of power will be complete; and in cases in which there are indications of considerable damage to the cord, it is almost certain that some lasting weakness will be left.

The electric irritability of the muscles and nerves may be expected to return to the normal more slowly than their capacity of response to the will, but sooner than their nutrition. The conditions that are observed in these cases are the same as in the isolated form of neuritis, in which, although the primary change is in the connective tissue, the symptoms depend on the secondary changes in the nerve-fibres. Hence the statements made on p. 69 are equally applicable to multiple neuritis. The reactions often furnish important information, espocially soon after the onset; the more the irritability deviates from the normal the greater will be the subsequent wasting and the longer the duration of the palsy. When some faradic irritability returns in both nerve and muscle, it may be regarded as a sure indication of advancing restoration of structure, which is certain to involve a progressive increase of conduction of voluntary impulse, and response to it. If the trunk nerves are not involved, the prognosis in severe cases is a little better when the arms are chiefly affected than when the legs have suffered most.

The sensory disturbance may be expected to follow a course, on the whole, similar to that of the motor symptoms. A diminution in the spontaneous pain may be anticipated sooner than in the tenderness of the nerves and muscles, which usually lasts, in lessening degree, until the muscles have regained some power. The extreme tenderness, indeed, passes away with the spontaneous pain, but some degree of over-sensitiveness continues for a long time. Happily the tendency to persistent neuralgia, seen after the isolated form, need not be anticipated after multiple neuritis. We find the explanation of this in the absence of the new formation of connective tissue from the inflammatory products, which in the isolated form perpetuate, by cicatricial contraction, the irritation to which inflammation gave rise.

There is little difference in the prognosis as regards recovery, in the different forms of neuritis. The anticipations that may be formed are governed in all by the rules just mentioned, and the risk to life has been already considered. In all, however, it may be repeated the extent to which the cause is under control forms an essential element in the problem, and must influence the conclusion in every case. The prognosis, however, is better in the sensory than in the motor form, better when the arms escape than when all the limbs are involved, and better in cases of chronic than of acute onset, and better if an apparently acute onset is really such than if it succeeds slight symptoms of longer duration. When the malady follows an acute specific disease, the severity of the latter is no guide to the course of its sequel; severe neuritis may follow a mild attack of the acute specific.

Treatment.—The different classes of multiple neuritis here considered need treatment that in some respects varies, and in others is the same in all forms. In all it is essential to discover, if possible, the cause of the neuritis, and to remove it or arrest the continuance of its action. This obviously should be the first consideration in cases due to alcohol, in which it is sometimes of great difficulty. It is desirable, as a rule, that alcohol in all forms should be given up entirely, but often this has to be done by degrees, a great reduction in amount preceding entire abstinence from it. With patients who are "their own masters" the reduction is often most difficult to secure, and it is necessary to give a warning that not only will life be in danger, but that the pains will certainly increase, and continued indulgence will entail sufferings so great that any sacrifice would be made, or deprivation endured, if their intensity could be realised. With every female drinker, deception should be considered not only possible but probable; those who are under the control of relations or of a husband, have generally had a long training in duplicity in order to procure stimulants, and it is easy for them to continue it, even when kept in bed, if they are attended by their own servants. These are corrupted, and alcohol is taken in secret when the fact is being stoutly denied. If a case does not present the arrest or improvement that might reasonably be expected from the withdrawal of the cause, such continued drinking should always be suspected. Hence it is of great importance that such patients should be, whenever practicable, under the immediate care of trustworthy trained nurses, rather than of their own servants or of relatives who may be improperly influenced or persuaded; if the patient's means do not permit this, removal to a hospital or some public institution is desirable. When the heart is too weak to permit the entire withdrawal of stimulants, the amount taken must be regulated solely by the state of the pulse. In such cases total abstinence is attended with some danger of the cardiac failure which has been mentioned as one of the causes of death. When the disease is due to cold, the patient should be carefully preserved from risk of further exposure, which, during the early stages, may convert a slight into a severe attack. Even in alcoholic cases, a chill will cause a rapid increase in the symptoms, and both this and fatigue should be carefully guarded against. In other forms, also, these measures, adopted in the stage when pains and tenderness are the only or chief symptoms, would often prevent the further development of the disease.

Physical rest is of great importance, and in all cases, except the slightest, rest in bed is desirable. The excitation of the nerves caused by movement tends to intensify the morbid process in them, and so does whatever produces pain. If the patient is in bed, local treatment can be more readily employed, and the restraint facilitates the necessary control over habits. The feeding of the patient is an important part of treatment, especially when the stomach has been deranged by alcohol. At the outset, warm fomentations should be applied over any tender nerves. Care must be taken in their use over the region supplied by the affected nerves, on account of the danger of vesication; troublesome ulcers may be caused by even moderate degrees of heat, as in the case mentioned on p. 62. Such applications, and also counter-irritants, have less effect in the parenchymatous forms than when there is a tendency for the connective tissue and sheath to suffer, and for their vessels to be involved in the process. A warm bath, for fifteen or twenty minutes daily, is often of service if the patient is able to bear the necessary procedures.

Care should be taken that the patient does not habitually assume a posture that will promote the occurrence of deformities. The dropping of the feet is the greatest danger, and should be prevented by a large and heavy sand-bag about nine inches in diameter, which may be adapted to the position of the feet, or by a board placed across the bed. Another danger is the habitual flexion of the knees, permitting a contraction of the hamstring muscles, and due to the discomfort that is caused by extension of the knees; this should, however, be insisted on because the initial discomfort soon passes away if extension is adopted from the outset. The extension of the knees involves that of the hip-joint, and prevents contraction of its flexors.

No drug has any very marked influence on the morbid process. It has been seen that multiple neuritis tends to lessen after its cause has ceased to act, and under such conditions it is easy to come to the conclusion that drugs do good which merely do not hinder. Mercury, which is certainly useful in isolated perineuritis, has little influence on the parenchymatous form, but it may be tried in any case in which it seems probable that the sheaths are primarily affected, and in which pain and tenderness of the larger nerve-trunks is a prominent symptom. During an acute febrile onset the treatment should be that adapted to the general state, and must differ according to its cause. In such an onset of an alcoholic neuritis, citrate of potash, nitrous ether, and compound tincture of cinchona are suitable, with a little digitalis if there is feebleness of the pulse. When due to cold, salicylate of soda may be given, or, if the attack has been predisposed to by alcohol, salicylate of potash may be substituted so as to avoid the retention of uric acid in the system by the soda. The probability of a gouty state of the system should be remembered in all cases in which there is a history of alcoholism. In the toxæmic forms in which there is a suspicion of a blood-state allied to that of septicæmia, no drug affords such a prospect of influence as the tincture of the perchloride of iron in full doses, 20—30 minims three or four times a day. Afterwards, when the actual onset is over, and from the first in the more chronic cases, tonics answer best—iron, quinine, or small doses of strychnine. Iodide of potassium, for some reason, seems to do good only in the chronic sensory form. In this also arsenic has been found useful, but arsenic should be given with caution, and in small doses; there is some danger that large doses may intensify the malady, since polyneuritis is one of the effects of chronic poisoning by arsenic, and has actually been produced by the medicinal use of the metal. Cod-liver oil is useful in the later stages, especially if there is impairment of general nutrition. In alcoholic cases, the addition of cocaine, $\frac{1}{12}$ to $\frac{1}{4}$ grain to each dose of whatever is taken, aids in lessening the craving for stimulant. When due to malarial poisoning, quinine may be given freely. The complications of multiple neuritis do not need other treatment than that suitable to them when they occur alone, nor do they indicate any special modification in the treatment of the disease of the nerves.

In most cases the pain is such as to render anodynes necessary. Of these, morphia, by hypodermic injection, is the most effective, but should be employed only as a last resource, and so regarded, will not often be found necessary. Its danger is the readiness with which those persons who were previously alcoholic become habituated to it. It may be given by the mouth with less risk, but in other drugs we may find a substitute that is effective in most cases. Injections of cocaine beneath the skin over the seat of pain often afford considerable relief, and cocaine may thus be given instead of by the mouth, securing the double object of lessening at once the local pain and the craving for stimulant—which is relieved almost as effectually when cocaine is given by the skin as by the mouth. Atropine may also be given hypodermically. In antipyrine and acetanilide we have drugs of another nature, that often give great relief to spontaneous pain. Indian hemp may also be given regularly without harm, and without interfering with the occasional administration of other anodynes; while to procure sleep, if pain is slight, reliance may be placed on bromide, sulphonal, or chloralamide. Antipyrine also often gives a patient a good night in spite of some pain. When there is mental excitement, hyoscine is useful, especially by hypodermic injection in doses of $\frac{1}{400}$ to $\frac{1}{150}$ of a grain.

The application of electricity to the paralysed muscles is important in order to maintain their nutrition in as good a state as possible during the process of recovery of the nerves. Only voltaism has the power of doing this, and should be applied daily, large sponge-holders being employed so that the current may reach as much muscular tissue as possible; only such a strength should be employed as will produce visible contraction and cause no after-pain. Voluntary muscular action constitutes a more effective stimulus to nutrition; but if, although the muscle can be put in action by the will, there is loss or great diminution of faradic irritability, or an excess of voltaic irritability shows that some fibres are in an abnormal state in consequence of the degeneration of their nerves, voltaism may still be applied with advantage. There is no evidence that the application of electricity to the nerves has any influence on their regeneration. Slight sensory loss is sometimes lessened in the chronic stage by the application of faradism by the wire brush.

The tender limbs may be wrapped in cotton-wool, with or without a covering of oiled silk. Massage is of service in the later stages of the disorder, for its influence on the nutrition and circulation in the affected limbs. An upward movement of the pressing hand helps the circulation of fluid in the vessels and in the tissues. It cannot be borne during the acutely painful stage, and, indeed, as long as it gives pain, it probably has more capacity for harm than for good. In the later stages it helps to overcome the contracture of the muscles, which should be gently extended at the same time as they are rubbed, —pressure, for instance, being made upon the ball of the foot at the same time as the calf muscles are rubbed.

The very long course of all severe cases makes a heavy demand on the patience of the sufferer and the perseverance of the medical practitioner; but the prolonged convalescence has the advantage, in alcoholic cases, of enabling a habit of abstinence to be formed. This, with the recollection of what has been endured, renders multiple neuritis more often a cure of intemperance than any other of the many maladies to which alcohol gives rise. During the slow recovery, the measures above indicated—tonics, electricity, and massage—should be continued. As already stated, the power of standing is interfered with for a time after the muscles have regained adequate strength, by the contraction of the gastrocnemii, causing a degree of talipes equinus. This is often so considerable as to suggest the desirability of dividing the tendo Achillis, but the operation is seldom if ever necessary. The attempt to stand and walk constitutes a powerful means of extension of the calf muscles, before which they soon yield sufficiently to permit the balance of the body to be maintained, and then progress becomes more rapid. The contraction at the knee and hip is more difficult to get rid of, but generally yields in time to persevering and gentle efforts.

ENDEMIC NEURITIS.

The clear evidence that has accumulated, showing the dependence of multiple neuritis on toxic blood-states, some connected with an organismal virus, might prepare us to find that it sometimes results from such poisons of endemic character. It is probable that many varieties of neuritis of this origin will yet be discovered; at present it is proved to be part of only three such diseases,—the malaria of remittent fever, leprosy, and the malady so widely prevalent in certain regions of the earth, the Kak-ké of Japan, Beri-beri of the Eastern Archipelago.

Malarial Neuritis.—Several cases have come under my notice in which persons living in districts in which remittent fever is endemic have suffered from weakness in the legs, chiefly in the muscles of the foot, and far greater in the anterior tibial group of muscles—the flexors of the ankle and extensors of the toes—than in any others. In most cases the paralysis of these muscles was absolute, and they presented the reactions of degeneration. The hands were unaffected. Such palsy is typical of peripheral neuritis. In one case there was a suspicion of alcoholic excess, but in the others there was no history of this or of any other cause than the exposure to malaria. There was slight muscular tenderness in some, but the more acute symptoms had had an opportunity of passing away during the long sea voyage to England. In all the sphincters were unaffected. The symptoms steadily improved, quinine being given and the muscles stimulated by voltaism. Contraction of the calf muscles had, however, resulted from the foot-drop in all the cases, so that the foot could not be brought beyond or up to a right angle; but as soon as the patients were able to attempt to stand, this contracture, however great, gradually yielded, and all made a good recovery.

This outline embodies the chief symptoms of the disorder. It must be regarded as a peripheral neuritis, involving chiefly the motor nerves and confined to the legs, at least in cases of moderate severity. It is probably due to the malarial agent that causes the endemic fever of tropical countries—which is presumed to be of organismal nature —or to some virus left behind by this. Its occurrence may be aided by alcoholism or excited by exposure to cold, but no case came from a non-malarial district. How frequent it is we cannot at present say. Its nature had not been recognised in any of the cases that have come under my own notice, the symptoms having been ascribed to disease of the spinal cord. But most physicians who have practised in such districts as those in which it occurs (India, the East Indies, coast of Africa, &c.) have recollected such cases when the symptoms were described to them, and it may perhaps turn out to be far from rare, and to occur also in a more acute and general form, the nature of which has also been misunderstood. Its symptoms and pathology are of especial interest in connection with the variety next to be described.

Beri-beri, the **Kak-ké** of Japan, is also known by other popular names in the various countries in which it is native.* Probably many names and localities remain to be discovered, or rather identified, for it is a mysteriously wide-spread disease, having apparently its chief homes in Japan, the Eastern Archipelago, India, New Zealand, Ceylon, the South Pacific Islands, and the coast of Brazil. It is especially prevalent in the Dutch East Indies, among the soldiers and in the prisons, and this has led to its systematic investigation under the direction of the Netherlands Government. Through this our knowledge of the malady has been much increased, especially by the investigations of Pekelharing and Winkler,[*] who had a large number of cases under their observation both during life and after death. Opinion as to its nature and cause has varied much, and still is far from uniform among those who have studied it; but there is a strong preponderance of evidence that it depends on a specific organism, and that symmetrical peripheral neuritis is the common effect of the virus, and the mechanism by which its chief symptoms are produced. The organisms found[†] are in the form of rods and cocci, but it is probable that these are only different stages in the development of the same species. They have been cultivated, and peripheral neuritis of nearly the same distribution as in beri-beri has been produced by their inoculation. Repeated inoculations are, however, necessary for this result to be produced; and hence, from this and the phenomena of the disease, it is assumed to be not a simple infectious malady, capable of being induced by a single exposure to its cause, but one in which repeated opportunities for infection are necessary. In harmony with this are the facts that sufferers acquire the disease from residence in certain infected houses, or places in which it is supposed that the soil is saturated with the organisms; that they may rapidly recover on removal to a district that is free, and relapse only on returning to an infected place; that it prevails where persons are gathered together, as in barracks and prisons; and that the air of these places is found to contain the organisms which, collected from it, will cause the disease in animals, from whom it is transmissible to

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* Beri-beri is probably a modification of the Cingalese name for the disease, bahr

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bari = extreme weakness. Kak-ké is the old Chinese name for it, by which it is mentioned by their medical works since 200 B.C.; it is derived from two words, meaning "legs" and "disease." The affection disappeared from China two centuries ago, remaining in Japan, where it continues a most serious endemic malady.

[*] Pekelharing and Winkler, 'Onderzoek naar den aarden de oorzaak der Beri-beri,' Utrecht (sep. publication), 1889, analysed in the 'Centralbl. f. Nervenkr.,' 1889; also 'Weekblad f. Nederl. Geneesk.,' 1888, and 'Deut. med. Wochenschr.,' 1888, No. 30. Other important contributions to the subject are by Bälz, 'Zeitschr. f. kl. Med.,' 1881; Scheube, ib., 1882; Weintraub, 'Wien. med. Wochenschr.,' 1888, Nos. 23—44; Van Eecke, 'Tijdschr. v. Nederl. Indre,' 1887, p. 71; Wernich, 'Virchow's Archiv,' Bd. lxxi; Minra, 'Virchow's Archiv.' Bd. cxi and cxiv, whose conclusions, however, differ from those of most investigators; Seguin, 'Phil. Med. and Surg. Rep.,' 1888; and Springthorpe, 'Australian Med. Journal,' 1889. Bälz, in 1881, expressed the opinion that the disease was a specific "panneuritis."

[†] First described by Bälz and Scheube, and since by Pekelharing and Winkler, Eigkman, Weintraub, Springthorpe, and others.

others by further inoculation. The serious extent to which it may spread among those who live together under conditions favouring its extension, is shown by the instance of a ship arriving in Japan from New Zealand after a voyage of 272 days. The disease spread on board so rapidly that altogether 169 cases occurred, with twenty-five deaths. In 1878 no less than 38 per cent. of Japanese soldiers were affected. It is probable that the inhalation of dried organisms floating in the air as part of "dust," is the chief way in which the disease is propagated. By some it has been thought that a nitrogenous diet induces or predisposes to the disease, and facts apparently supporting the opinion have been adduced; an exclusive fish diet has been thought to cause it, and so also has a rice diet. A remarkable outbreak in Manila in 1880 followed a period of rice-eating during a cholera epidemic. These influences probably merely produce susceptibility.

It is a remarkable fact that Europeans seldom suffer. Males are more liable to the disease than females, and it is chiefly prevalent during the hot season.

Symptoms.—Peripheral symmetrical neuritis is a constant feature of the malady, which is usually chronic in course and gradual in onset, but prone to undergo acute exacerbations. These have been generally regarded as acute forms of the disease, but it is said by Pekelharing that the symptoms of neuritis may always be discovered before the onset of the definite symptoms, and this when the patient is ignorant of their existence and feels quite well. Most observers have failed to recognise this, and it is probably true only under certain conditions, and persons suddenly exposed to an intense infection may suffer acutely from the very commencement. Occasionally the malady develops with extreme rapidity and severity. On the other hand, many cases are chronic throughout, and last for months. The leading symptoms are those of multiple neuritis (chiefly affecting the legs and the cardiac branches of the vagus), dropsy, and symptoms of cardiac failure. The amount of urine is generally lessened, and its secretion may be almost suppressed in acute cases. A "critical" increase in the secretion may mark the commencement of improvement. It is unchanged in character, and does not contain albumen. The dropsy is a very variable symptom, and this has led to the distinction of two forms, the dry and the dropsical. Pekelharing and Winkler, however, found some effusion of fluid almost invariably after death, and it is probable that its conspicuous presence or absence depends chiefly on the state of the heart (conditioned by that of its nerves), and partly on the trophic and vaso-motor disturbances due to the local neuritis.

The earliest symptoms are a change in the electrical excitability of the peroneal nerves and the flexors of the ankles (which suffer most throughout)—a slight degree of the reaction of degeneration, quantitative and often qualitative. These are often to be found before there are any subjective symptoms, which begin as a sense of heaviness of the legs, readiness of fatigue, dysæsthesiæ and diminution of tactile sensibility in the lower legs, palpitation and undue excitability of the heart. The electrical changes may be met with in slight cases which proceed no further,* and have shown that some patients, supposed to be shamming, were real sufferers. To these subjective symptoms are added other objective signs,—œdema along the edge of the tibiæ, a peculiar pasty and stiff aspect in the face, an increase in the cardiac dulness to the right, roughness of the first and accentuation of the pulmonary second sound. These symptoms may increase slowly, or rapidly in the form of an acute stage. The degenerative reaction becomes complete in the muscles first affected, and they become paralysed and undergo the characteristic atrophy, while other nerves and muscles progressively suffer in the same manner, the calf muscles, the extensors of the knee, the adductors of the thigh, and, lastly, the flexors of the thigh and the abductors of the hip. In the trunk the abdominal muscles and intercostals may be involved, and in severe cases the arms are paralysed, first the extensors of the wrist and fingers, later the flexors, and sometimes most of the muscles may be so atrophied as almost to disappear. The face often suffers: the diaphragm may become paralysed, and also the larynx (the inferior before the superior laryngeal nerve); while grave cardiac weakness and increasing dilatation of the heart testify to the serious implication of the cardiac branches of the vagus. Simultaneously, sensory symptoms develop, corresponding in distribution to the more severe motor symptoms; sensibility to touch is lessened or lost (first on the inner side of the lower leg), while that to pain usually remains, and sometimes is augmented to the degree that constitutes what has been termed "anæsthesia dolorosa." The temperature-sense may be diminished to either heat or cold or both in various parts, and there is often considerable loss of cutaneous sensitiveness to faradism. There may be tingling, formication, and other dysæsthesiæ, together with tenderness of the nerves and muscles, but far less in degree than in most forms of polyneuritis, and chiefly marked in the early stage of the disease.

The œdema that is so common begins in the legs, usually spreads widely, and involves not only the subcutaneous tissue but the cavities of the peritoneum, pleura, and pericardium: in the last it seriously impedes the action of the already dilated and feeble heart. It is to the cardiac failure that death is commonly due; the other chief cause is failure of the respiratory muscles, aided generally by effusion into the pleural cavities, and accompanied by the indications of cardiac weakness, and of dilatation of the right side of the heart. From this cause the cardiac dulness sometimes increases rapidly, even in a few hours. The œdema is usually attended by increasing anæmia, which doubtless facilitates its occurrence. These symptoms are always later in development than are those due directly to the neuritis. In the most acute cases the definite symptoms take commonly six or eight weeks to run their course, and the usual duration of the malady is several months. Death from the heart failure is usually prolonged, and accompanied by much distress. Occasionally a very acute form prevails, in which rapid failure of the heart and various effusions lead to death in about a fortnight, and cause a mortality of 60 and 70 per cent.

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* Pekelharing and Winkler, Eigkman (1889), &c.

Pathological Anatomy.—The only constant changes are in the nerves, but some serous effusion is almost invariable. Thus Pekelharing and Winkler found an excess of fluid in the cellular tissue in 83 out of 85 cases—about 97 per cent. The pericardial fluid was increased in 67 per cent. of the cases, in about 23 per cent. there was hydrothorax, and ascites in 14 per cent.

The changes in the nerves are often indistinct to the naked eye, although always conspicuous under the microscope. They are met with in both motor and sensory branches, and, in advanced cases, in the nerves of the vessels. They are always greatest at the terminal portions, lessening upwards, while the nerve-roots and spinal cord are always normal, with the doubtful exception of vacuolation of the motor cells,[*] and some degeneration of the fibres of the posterior column in a few cases.[†] The first change is an increase in the segmental nuclei, shrinkage of the medullary sheath at the nodes, and a peculiar "lumpy" degeneration of the medulla, sometimes replaced by a "foam-like" appearance in it, concealing the axis-cylinder. Both changes are often accompanied by the formation of granule corpuscles. These slighter changes are found chiefly where the lesion is commencing, and especially in the vagus and phrenic nerves, in which, necessarily, a considerable degree of change is incompatible with life. They have been seen in the branches within the heart, in some of the fibres of the trunk, and in the laryngeal nerves. Where the change in the nerves is more advanced, the white substance is entirely broken up, and the axis-cylinder presents segmentation or irregular swellings, or has disappeared, and the increase in the nuclei of the neurilemma sheath is still greater than before. The blood-vessels of the nerves are but little changed in the early stage; after a time they may present some thickening of the wall, accompanied by a general increase in the amount of the interstitial tissue; but, as a rule, there is an absence of any signs of interstitial inflammation—the changes are essentially parenchymatous.

In the muscles there may be at first a cloudy opacity with indistinctness of the transverse striæ, going on to granular degeneration; or the fibres may simply become narrow, preserving their striation. The nuclei of the fibre-sheath are always increased in number, but the interstitial tissue is generally but little altered.

Pathology.—The characteristic organisms have been invariably found in the blood, and, what is a remarkable instance of their slow action, and of the cumulative influence needed to generate the malady, Pekelharing and Winkler[*] found them in the blood of all the inhabitants of an intensely infected place, whether these had or had not symptoms of the disease, and failed to find them in the blood of persons living in places free from infection. Cultures from the blood reproduce the malady, and so do cultures from the animals thus infected. The organisms have not been found in special association with the morbid process in the nerves. It thus appears that the malady bears a close resemblance, in its essential features, to cases of multiple neuritis due to a chronic blood-poison such as alcohol, and still more to that which, as we shall see, is presented by chronic arsenical poisoning. There is a slow accumulation in the blood of a virus that has a specific action on the peripheral nerves, and like some others, first and chiefly on the nerves of the legs, but with a greater affinity for the vagus, especially for its cardiac branches, than most others. Although invariably associated with the presence in the blood of a special organism, the absence of this in the vicinity of the lesion, taken in conjunction with modern researches in bacteriology, makes it probable that the virus is a chemical substance produced by the organism in its growth. This brings the disease into close affinity with some other forms of multiple neuritis, on which it throws sufficient light to justify this somewhat full account of a malady that is not likely practically to concern many readers. If we regard, as apparently we must, the cardiac failure as the result of the cardiac neuritis, and the dropsical effusion as the combined result of this and the local trophic and vaso-motor influence of the neuritis (to which, perhaps, the change in the blood should be added), we have a remarkable illustration of the extent, variety, and degree of the secondary processes of disease which multiple neuritis may produce.

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[*] Miura, loc. cit.

[†] Van Eecke loc. cit

The *Diagnosis* can only be a matter of difficulty in a very early case, or in a patient who is not known to have been exposed to the infection, or in consequence of ignorance of the disease. Doubt may exist, however, whether in a given case the malady is alcoholic neuritis or beri-beri. The former is probable if the arms are involved at the same time as the legs, still more if they suffer first; also if pains are severe, œdema absent, and the heart unaffected. The converse would make the endemic malady probable. Beri-beri has certainly been intensified by alcohol in some cases, and the symptoms then presented are a combination of the two,—pains, associated with the weakness and enlargement of the heart and the wide-spread tendency to dropsical effusion met with in beri-beri. In most cases some other indication of the influence of alcohol, especially enlargement of the liver, will assist the diagnosis. From other forms of multiple neuritis it is unlikely that the diagnosis would have to be made. From affections of the spinal cord the distinction (chiefly needed in the chronic "dry" variety) depends on the points already mentioned in the account of multiple neuritis. Acute cases with much œdema and weakness of the heart, and enlargement of the right side, may be mistaken for primary heart disease; but if the symptoms of beri-beri are known, the affection may be recognised by the absence of any indications of primary valvular disease; while an examination of the legs will reveal the weakness of the flexors of the foot and lessened tactile sensibility in the skin over them.

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[*] Also Ogata (1888) and others. They have also been found sparingly in the organs, even in the spinal cord, which is commonly free from disease.

Treatment.—Beri-beri is one of those diseases for which it is probable that a method of at least prophylactic inoculation will be discovered in the future. At present the destruction of the organisms by disinfecting agents, or, better, the removal of a person to an uninfected district, constitutes the only means of preserving him from the continued entrance of the poison—a measure essential for successful treatment, and, in many cases, all that is really necessary. The limits of disinfection are, however, reached with the dwelling-house; and the great difficulty presented by the disease is the manner in which whole districts are capable of giving rise to it, probably by the infection of the soil, which cannot be rendered innocuous. However, even within the narrow limits of personal use, disinfection has achieved some remarkable results, and in few diseases has the light of pathology given more useful guidance to the hand of the practitioner.

In most respects the treatment of the symptoms of beri-beri does not differ from that of alcoholic neuritis. A special point, however, is that the strengthening of the heart by digitalis or strophanthus is more often of urgent importance, and requires greater care lest the feeble fibres be overtaxed. Small doses should therefore be given—three or at most five minims of the tincture of digitalis. Bodily rest is of paramount importance to the weak organ, in order that there may not be any demand on its energies for other work than that involved in the maintenance of what may be termed static nutrition, and that it may be spared the increased work necessitated by muscular exertion. Strychnia, quinine, &c., may do good, but their use in this disease does not differ from that in the more common forms of multiple neuritis.

LEPROUS NEURITIS.

Far removed as leprosy may seem to be from the malady last described, both by its general character and extremely chronic course, it is certainly also an endemic disease; its cause must be sought in local influences; and it agrees with beri-beri in the nature of that influence. Since the researches of Carter, Hansen, and Neisser have proved the constant presence of a specific bacillus in all the new formations met with in the disease, their observations have received abundant corroboration; the disease was indeed one of the first to be proved to depend on living organisms. The virus resembles that of beri-beri in being transmissible from individual to individual, but differs in its capability of passage by inheritance, in the less readiness with which it passes from person to person, and in the extreme slowness with which it commonly develops to the degree necessary to cause symptoms. Years may elapse after the last possible exposure, before distinct symptoms of leprosy appear. In the case from which Fig. 62 was taken the sufferer was a European of healthy parentage, who could not have been infected within ten years of the first symptoms. In one case of infection from adult to adult, nine years elapsed after the death of the leper before the man who had lived with him began to suffer. When, indeed, a large quantity of the virus is received, the malady develops much more rapidly. But leprosy differs from beri-beri, and from most other forms of multiple neuritis, in the fact that although inflammation of many nerves is frequently a part of the disease, it is not an essential part or a constant feature of the affection. On the contrary, it is only one of an extensive series of lesions, inconstant, not only in its occurrence, but in its seat and extent. It characterises a special variety of the disease, the "anæsthetic leprosy" in which not only areas of the skin become insensitive, but the ends of the fingers may suffer such an impairment of their nutrition that they are lost. Moreover, as we have seen in the introductory section, the want of strict symmetry is associated with an important difference from the symmetrical forms—the neuritis is not a parenchymatous or degenerative form, but is a perineuritis and interstitial neuritis, and the damage to the fibres is secondary. This change in the connective tissue is associated with the actual presence of the organisms in it. The neuritis thus seems to be due to the direct action of the bacilli on the affected tissues, and not, as in beri-beri, to a virus circulating in the blood—not to a product of the organisms, but to the organisms themselves. This fact enables us to understand better its peculiar features, and especially its irregular distribution; the inflammation occurs where the organisms happen to fix themselves in the connective tissue, and although certain common conditions on the two sides may determine a partial correspondence in the nerves affected, there is not the symmetry that results from an action on like structures on the two sides, equally accessible to the virus. In this, and in its connective-tissue seat, the neuritis of leprosy resembles the more isolated neuritis of syphilis, rather than the common varieties of the multiple form.

The symptoms of the malady are extensive and various, but only those that consist in derangement of the functions of the nervous system concern us here, and it is alike needless and impracticable to describe the others. The nervous symptoms of leprosy depend almost exclusively on inflammation of the nerves. These symptoms are muscular wasting and anæsthesia, greatest in degree towards the extremities of the limbs, but more or less irregular in situation and in the region of the distribution of nerve-trunks, which, if accessible, are obviously diseased. The symptoms are not confined to the limbs, but are also met with in the face, where an affection of the fifth nerve may cause anæsthesia, and movement may suffer from involvement of the trunk of the facial nerve, so that the eyelids cannot be completely closed. The form of sensibility that is impaired also varies; sometimes touch, sometimes pain is lessened in greater degree, but usually all forms are implicated to some extent. The legs often suffer before the arms, but a special order of affection cannot be distinguished. There may be unpleasant tingling sensations, but pains are usually trifling, perhaps because the process is extremely chronic. In some cases there may be severe pain in the position of the nerves or deeply seated in the limbs. The inequality of the affection of the fibres of the same nerve is shown by the fact that the knee-jerk may persist although the extensors of the knee are partially paralysed. Fig. 61 shows the distribution of anæsthesia in the hands in a well-marked example of this disease which came under my observation some years ago. The patient was a creole of Mauritius, who had come to England in early childhood, and presented the first distinct symptoms at twelve years of age—changes in the pigmentation of the skin, anæsthesia, and muscular wasting. They steadily increased during the next four years, the anæsthesia extending each half-year from one finger to another. There was also scattered anæsthesia of the legs below the knee. As the figure shows, the loss of sensation does not correspond in area to nerve distribution. This depends on the fact that even when all the nerves, including those of the fingers, are diseased, all the fibres may not be destroyed in any one nerve, so that some sensibility exists in each nerve area. Various perversions of sensibility, spontaneous "dysæsthesiæ," tingling, formication, and the like, may precede the loss. The latter usually occurs gradually, but sometimes so suddenly as to suggest some secondary vascular lesion, hæmorrhage or thrombosis, in the vessels of the nerve that is the seat of the process—a possible result in its

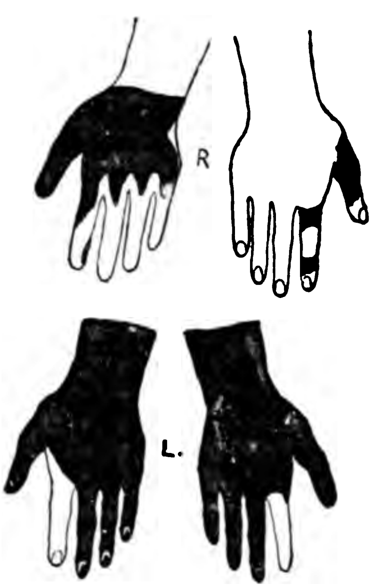

FIG. 61.—Distribution of anæsthesia in a case of leprosy. The black areas indicate the loss.

early and most active stage. It must be remembered that in all inflammations of the sheath and interstitial tissues this proceeds to a considerable degree before the fibres of the nerve suffer, and thus sudden loss of sensibility does not show that the nerve was previously healthy. This occasionally sudden onset is of much practical importance; one case was sent to me from a place in which the disease is endemic, by a doctor well acquainted with it, who had not suspected leprosy because the anæsthesia came on almost suddenly.

Accompanying anæsthesia there is usually some muscular wasting, which may be great. In the case figured it was considerable, although only in the small muscles of the hands was it comparable to that of progressive muscular atrophy. The electrical excitability of the muscles was greatly lowered to each current, and this is perhaps the most common condition; either the damage to the nerves is so chronic that the fibres undergo slow changes *pari passu* with those in the nerves and their endings, or the patient comes under observation at a late stage in the local affection, at which the changes present in the early stage have disappeared; sometimes, indeed, all irritability has vanished. In cases that are seen soon after the onset of the lesion of the nerves, there may be the reaction of degeneration in the muscles, either partial or complete, and there is often a conspicuous exaltation of the mechanical irritability of the nerve-trunks. When the facial nerves are affected this may be well seen, and may resemble that met with in tetany.* The thickening of the nerve may be felt when that which is affected is accessible. The state of myotatic irritability varies according to the distribution of the neuritis; it is lost at places where the sensory or motor fibres are involved, but those supplying the front of the thigh and its muscles often escape, and the knee-jerk is preserved. Anchylosis of the joints may occur, as in other forms of neuritis.

The "mutilations" of leprosy, by which the ends of the fingers and toes are lost, are also regarded by some as a consequence of the neuritis. The numerous other symptoms of the disease are independent of the nerves (except perhaps the pigmentation of the skin), and are beyond the province of this book.

The neuritis of leprosy is typically adventitial (Fig. 62). The primary sheath and the secondary sheaths of the fasciculi are greatly increased in thickness, and consist of nucleated fibrous tissue arranged concentrically (A, B). From the sheath, tracts extend into the interior of each fasciculus (B), isolating the groups of nerve-fibres. The increase of tissue even extends between the fibres themselves (C), and these undergo slow wasting; many of the fibres in the figure are seen to be distinctly narrower than normal. The concentric growth of fibrous tissue may even invade the whole area of the fasciculus, all the nerve-fibres perishing before it (D). The characteristic bacillus of leprosy is found abundantly, in recent cases, in the new tissue of the nerves. Peculiar cells are met with, infiltrated with the organisms.

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* Schultze, 'Deut. Arch. f. klin. Med.,' 1888, Bd. xliii.

As the fibrous tissue develops and contracts, the bacilli seem to perish, and ultimately can no longer be discovered.

The diagnosis depends on the occurrence of irregular areas of anæsthesia, generally associated with irregular patches of pigmentation and pallor in the skin, and often with muscular atrophy, in a person who has been exposed to the risk of infection, generally by having lived in a district in which the disease is endemic. The irregularity of distribution and the limitation of considerable sensory changes sufficiently distinguish it from other forms of neuritis. The spinal affections with which the mixed anæsthesia and wasting are most likely to be confounded are, as Schultze has pointed out, cases of syringo-myelia in which the distension of the central cavity damages the grey matter, and may cause similar symptoms of irregular distribution. But these are limited to the arms; and if the legs suffer it is in a different way—as simple paraplegia with excess of myotatic irritability that may go on to spasm.

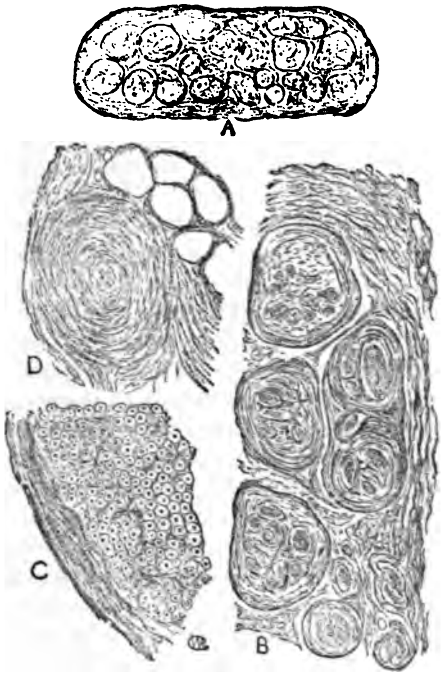

FIG. 62.—Sections of nerves from a case of anæsthetic leprosy, under the care of Dr. Buzzard. A, median nerve at wrist × 5; B, portion of same more highly magnified; C, part of a less diseased fasciculus from the ulnar nerve; D, a small fasciculus from median in which the concentric fibres have invaded the whole area of the fasciculus.

The prognosis of the disease in the simple anæsthetic form is grave only when the sufferer is still exposed to fresh infection, or in cases of considerable severity. But it must be remembered that, just as the disease may develop long after exposure to its cause, so it may slowly increase for a long time after this exposure has ceased.

The treatment of the affection is beyond the scope of this work; in so far as the nerve symptoms require special measures, these are the same as in ordinary neuritis. A trial may be made of the stimulation of the muscles by whatever form of electricity they will respond to, and of the anæsthetic areas in the skin by the wire brush and faradism.

PART III.

DISEASES OF THE SPINAL CORD.

INTRODUCTION.

ANATOMY OF THE SPINAL CORD.[*]

THE spinal cord, it will be remembered, is much shorter than the spinal canal, reaching only to the second lumbar vertebra. Hence the nerve-roots descend to their foramina of exit. The lower they arise, the longer is their intra-spinal course. All those below the second lumbar pair leave the canal below the lowest portion of the cord. It is customary to speak of the portion of the spinal cord from which each pair of nerves arise as the corresponding "segment" of the cord. The segments are longest in the dorsal region, and shortest in the lumbar enlargement. They are also called "metameres."

The only parts of the spinal column that we can usually feel are the vertebral spines. Many of these are not on a level with the bodies of their vertebræ. It is important, therefore, to know the relation of the spines to the bodies of the several vertebræ, and of these to the origin of the nerves. These relations are shown in the accompanying figure (Fig. 63).

Of the *Membranes*, the pia mater closely invests the surface, and is continuous with the tracts of connective tissue that pass within the substance of the spinal cord. It is also prolonged along the nerve-roots as their sheaths. The arachnoid forms a much less close investment. The dura mater is not in contact with the bones, as it is in the cranium, but a layer of fat and a plexus of large veins intervene between the two. It thus forms a loose sheath for the cord, and variations in the amount of blood in the plexuses outside it permit corresponding (but inverse) variations of the amount of cerebro-spina fluid within it. An extension of the dura mater passes along each nerve-root and blends with its sheath.

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[*] The following outline of the anatomy of the spinal cord is intended merely to place before the reader those points that are essential, or likely to become so, for understanding the functions of the organ and the symptoms of its disease. It does not profess to be exhaustive even in outline, and is designedly kept as free as possible from whatever, in either the substance or terminology of recent science, does not come within the scope of those objects. Many of the results reached by modern investigators are mutually incompatible, and where a choice has been necessary the observers have been followed who possess the greatest authority and whose conclusions best agree with facts previously ascertained.

STRUCTURE OF THE SPINAL CORD.—The general form of the cord, the enlargements it presents, and its constitution of white and grey substance, are too well known to need description here. The *white substance* surrounds the grey, except at the two points at which the posterior horns come to the surface (Fig. 64). It consists of medullated nerve-fibres, chiefly longitudinal. The posterior cornua isolate the posterior columns from the rest of the white substance. These columns are separated by a "posterior median septum" of connective tissue, and a little distance from this another incomplete septum, "posterior intermediate septum," corresponding to a depression on the surface, marks off a portion next the median septum, the "postero-median column," or "column of Goll," from the part next the posterior horn, "postero-external column," or "column of Burdach," the part of which next the cornu is called the "posterior root-zone," because many fibres of the

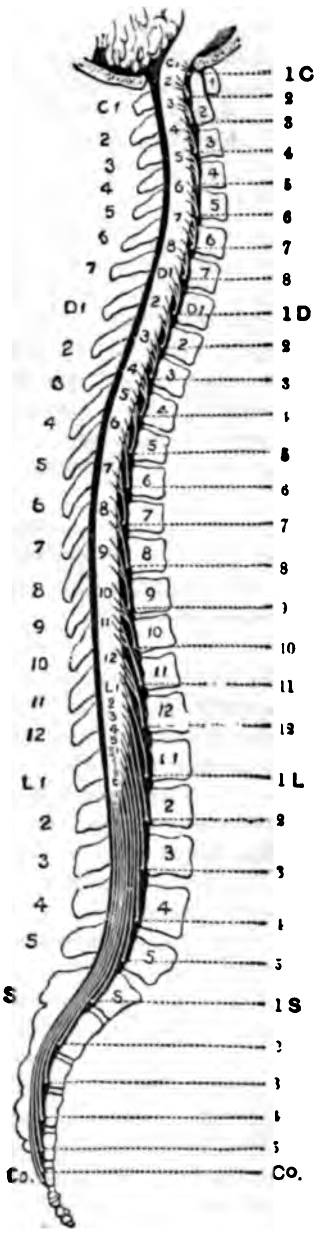

FIG. 63.—Diagram (framed from an original investigation) showing the relation of the vertebral spines to their bodies and to the origin of the several nerve-roots. It will be seen that the ends of the vertebral spines are opposite the middle of their own bodies only in the lumbar region; they correspond to the lower edge of their own bodies in the cervical and the last two dorsal vertebræ, and to the upper part of the body below in the rest of the dorsal region. Each cervical spine is nearly opposite the lower roots of the nerve below; the vertebra prominens is opposite the first dorsal roots, and from the 3rd to the 10th dorsal the spines correspond to the second root below; the 11th spine corresponds to the 1st and 2nd lumbar nerves, the 12th to the 3rd, 4th and 5th; the 1st lumbar to the 1st, 2nd and 3rd sacral nerves, while the top of the cord is opposite the upper part of the 2nd lumbar.

posterior root pass through it. The rest of the white substance is divided, in the middle line in front, by the anterior median fissure,

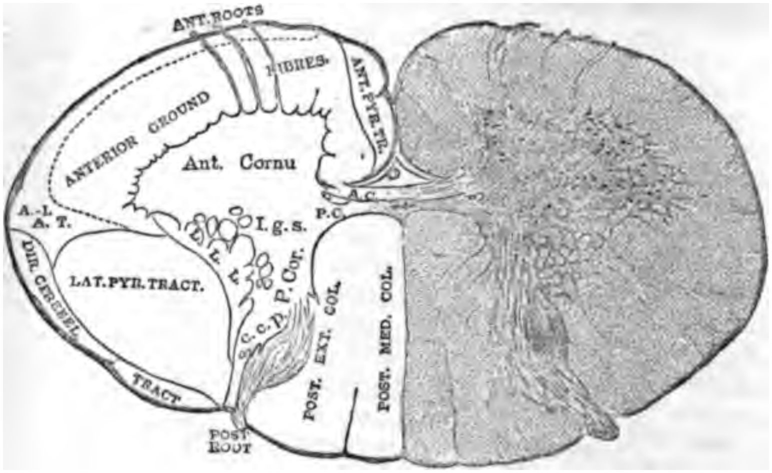

FIG. 64.—Diagram of a section of the spinal cord in the cervical region. A. C., anterior commissure; P. C., posterior commissure; I. g. s., intermediate grey substance; P. cor., posterior cornu; C. c. p., caput cornu posterioris; L. L. L., lateral limiting layer; A.-L. A. T., antero-lateral ascending tract, which extends along the periphery of the cord.

down which the pia mater and blood-vessels pass, and at the bottom of which is the anterior or white commissure. Between the anterior median fissure and the posterior cornu the white substance is continuous and undivided, extending round the front and side of the cord. It is artificially divided into an anterior and a lateral column, the line of division being the outermost of the anterior nerve-roots, which pass through the front of the cord; but there is no corresponding distinction of structure, and hence it is often termed the "antero-lateral column."

The white substance varies in amount in different parts of the cord, but, as a whole, lessens gradually from above downwards (see Fig. 65). It is everywhere composed of medullated nerve-fibres which, however, possess no neurilemma-sheath. In carmine-staine sections the axis-cylinder is seen within each fibre, not always in tl centre, and around this are concentric cloudy lines due to the irregul refraction of the white substance.

Between the fibres is a peculiar substance, the "neuroglia" nerve-cement. It appears to consist of fine fibres, which form a r work, embedded in a finely granular or homogeneous matrix. their intersections are peculiar cells consisting of a nucleus small cell body ("glia-cells," "cells of Deiters"). The fibres generally regarded as their processes, but, according to Ranvier

Weigert, the cells are flattened, without processes. This neuroglia is developed from the same (epithelial) embryological elements as the nerve-structures—an important fact, because it enables us to understand the fact that, in defective development of the nerve-structures, the neuroglial tissue is excessive in amount. But its differentiation in development involves also its chemical nature, since it consists of material like that of hair or horn, and hence called "neurokeratin."

The nerve-fibres, thus connected, lie in a coarser network, formed by branching processes of septa that proceed inwards from the pia mater. In these septa blood-vessels run, and some are occupied by nerve-fibres that have a horizontal course. The septa consist of fine fibres from the pia mater and of neuroglial material, which forms also a layer between the membrane and the nerve-fibres in the intervals between the septa. This layer varies considerably in thickness. Many of the septa pass through the white substance to join projections from the grey substance, which cause the irregularity of the outline of the latter. These processes, outside the neck of the posterior cornu, are broad, and join each other so as to form a sort of network, the meshes of which enclose columns of nerve-fibres. This is called the "lateral reticular formation" (Fig. 64, right side; also 65, c, 2—8). The substance of the horn behind the caput is sometimes broken up in the same way—the "posterior reticular formation."

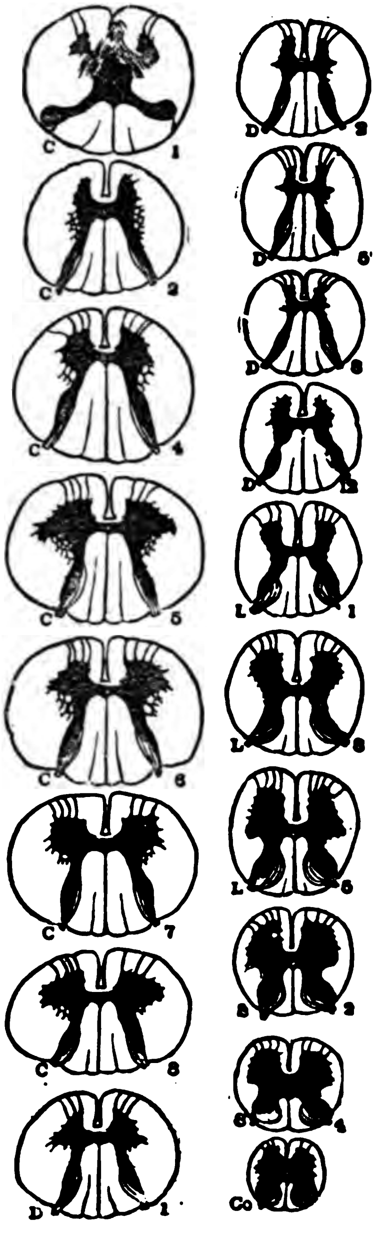

FIG. 65.—Diagram showing the relative size and shape of the cord and grey matter at different levels.

The *grey substance* varies in shape and size in different parts of the cord, being largest in the cervical and lumbar swellings, and corresponding to the number and size of the nerve-roots given off. The variations in shape and size are indicated in the accompanying figure (65), and will be readily understood by an examination of this. The division into anterior and posterior horns or cornua is familiar. The part on each side that intervenes between the two horns may be conveniently termed the "intermediate grey substance" (I. g. s. in Fig. 64). In the dorsal region a projection from this extends into the lateral column (Fig. 65, D 2, 5, and 8), and has been termed the "lateral horn"). It is customary to divide the grey matter into two varieties, "spongy" and "gelatinous." The latter forms a cap on the posterior horn, and a layer immediately around the central canal, and is named on account of its naked-eye aspect. The spongy substance, which forms the rest of the two cornua, consists mainly of an excessively fine felty network of very narrow medullated nerve-fibres, naked axis-cylinders, and fine nerve-fibrillæ which arise, in part at least, from the branching processes of the nerve-cells, and perhaps in part by a division of the axis-cylinders of the nerve-fibres. It also contains small cellular bodies, which vary in size and shape. Many are angular with processes, and are probably of nervous function. These structures are embedded in a supporting neuroglia. Through it course many larger medullated nerve-fibres, passing to or from the white columns and nerve-roots. In it also lie many nerve-cells of various sizes. Those in the anterior cornu are, for the most part, large "ganglion-cells;" each contains a large nucleus, and usually a mass of pigment, and sends out several processes. One process is continuous with the axis-cylinder of a nerve fibre (Fig. 66), and can be sometimes traced into a fibre of an anterior root (Fig. 67). The other processes, after a

FIG. 66.

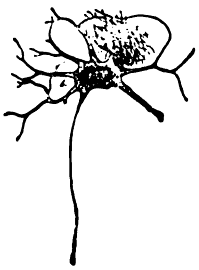

FIG. 67.

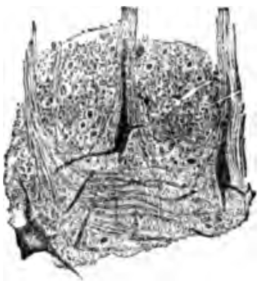

FIG. 68.

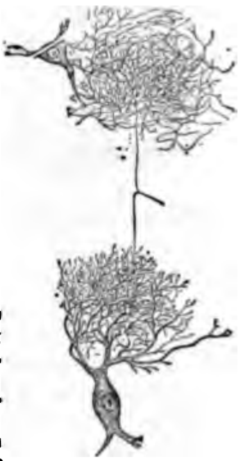

FIG. 66.—A nerve-cell from the anterior cornu of the spinal cord of man: *a*, unbranched process becoming the axis-cylinder of a nerve-fibre; *b*, pigment mass. (After Gerlach.)

FIG. 67.—Nerve-cells of the anterior cornu sending a process into the anterior root. (After Henle.)

FIG. 68.—A fine nerve-fibre dividing into two parts, each of which joins the plexus of fibrillæ formed by the branching processes of a nerve-cell. From the spinal cord of an ox. (After Gerlach.)

longer or shorter course, divide and subdivide, their ramifications being lost in the sponge-like matrix of the grey substance. In this they are probably connected with the terminal ramifications of nerve-fibres (Fig. 68). These cells lie in groups, between and through which course tracts of fibres, chiefly from the anterior roots. Other fibres come from the anterior commissure, white columns, and posterior horn. The ganglion-cells are certainly motor in nature, and give origin to at least most, and perhaps all, of the motor fibres of the spinal nerves. Birge, in a careful investigation, found that in each segment of the spinal cord of the frog the number of nerve-cells in the anterior cornu nearly corresponds to the number of fibres of the anterior roots arising from that segment. Each cell is traversed by many nerve-fibrillæ, such as are discerned in a cell-process or in the axis-cylinder of a nerve-fibre. These cross and intermingle in the middle of the cell, but pass along the margin from one process to another. By this arrangement impulses entering the cell by one process may leave it by many.*

The arrangement of the nerve-cells of the anterior cornu is of some practical importance, on account of the frequency with which this part is the seat of limited lesions. They form, as already stated, certain groups, but these groups vary in different regions of the cord, partly under the influence of the shape of the horn, partly irrespective of this, and they even vary in parts of the cord that are near together. They are influenced by the course of the tracts of fibres of the anterior roots, which may pass through a group and break it up into smaller groups, although in a neighbouring section it is undivided (see Fig. 69, the cervical groups). Hence very different descriptions have been given of these groups, and the process of distinction has sometimes been carried too far. The groups that can most readily be recognised are the following:—In the inner anterior angle of the cornu is a small group, the *inner* or *median group*. This is one of the smallest, and is absent in some parts of the cord, especially in the lumbar region. A much larger group lies near the anterior edge of the horn, in the middle, or a little to the outer side of the middle, the *anterior group*. External to this, in the outer extremity of the front part of the horn, is another group, the *antero-lateral group*. These two groups are often blended, so that the anterior group cannot be separately distinguished, as in the right-hand mid-cervical figure. A fourth group, usually the largest, lies in the outermost part of the horn, behind its front, usually in the posterior outer angle; it may extend inwards, halfway across the horn. It is called the *external* or *postero-lateral group* (P.-L., Fig. 69). These three are the most important groups.

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* Kronthal, 'Neur. Centralbl.,' 1890, p. 40. Impulses may thus be diffused, even if there is no junction of fibrillæ or passage of impulses from one fibril to another. These fibrillæ are said to be composed of fusiform elements, united at their extremities by finer threads of the substance of which they consist. They break down, two or three hours after death, into rows of fine granules. This arrangement provides a mechanism by which the nerve-cells may conduct and diffuse impulses, but such a mechanism does not exclude (as has been assumed) the reinforcement or origination of such impulses, and their discovery leaves untouched the facts which demonstrate the trophic influence of the cells, and the manner in which they arrest secondary degeneration. These functions are indicated by facts of a totally different nature. Kronthal's observations (by a new method) are of great importance, and appear trustworthy.

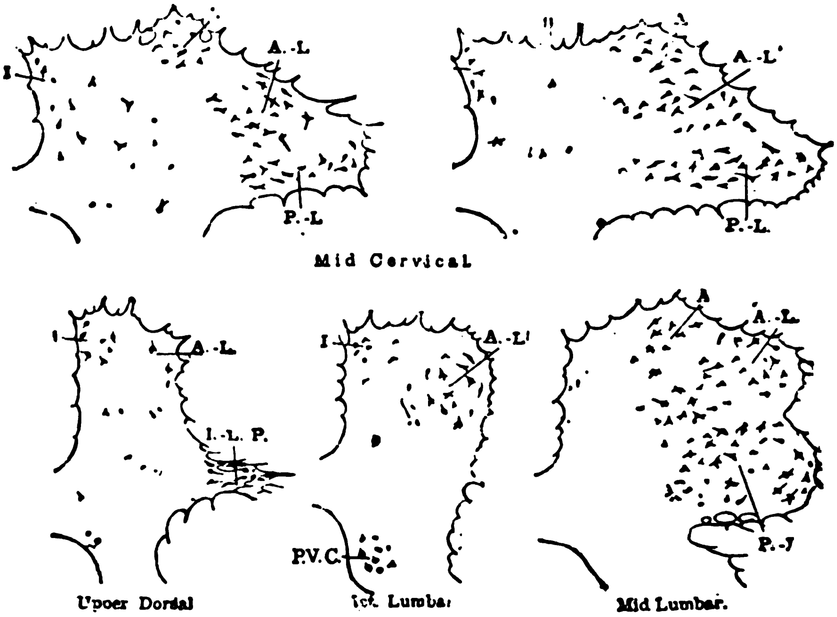

FIG. 69.—Diagrams of the groups of nerve-cells in the anterior cornu. Groups:—I., inner or medial; A., anterior; A.-L., antero-lateral; P.-L., postero-lateral; I. L. P., intermediate lateral process; P. V. C., posterior vesicular column or tract. The two mid-cervical sections are only a few millimetres apart, and show how the anterior group, separate in the one, may be blended with the antero-lateral group in a neighbouring part of the cord.

There is in some parts also a *central group*, occupying nearly the centre of the horn. In the small cornu of the dorsal region, often no well-defined groups can be made out, but when any can be recognised, they are generally the anterior and external.

Similar cells, usually smaller in size, and isolated, are scattered through the intermediate grey matter, and a group of cells occupies a projection outwards into the lateral column in the lower cervical and upper dorsal regions. It was termed by Lockhart Clarke the *intermedio-lateral tract*, but which is better designated the *intermediate process* (or *lateral horn*), and the cells the *intermediate group* (I. L. P., Fig. 69). Nerve-cells also lie in the grey matter of the reticular formations, lateral and posterior, and are sometimes described as special groups. Many isolated cells, medium and small in size, also lie in the posterior horn. These are probably connected with some of the fibres of the posterior roots. They extend back into the caput cornu posterioris.*

In the lower dorsal and upper lumbar cord, from the eighth dorsal to the second lumbar nerves, a group of nerve-cells lies in the inner part of the neck of the posterior horn, the *posterior vesicular column* of Lockhart Clarke (sometimes called "Clarke's column") (P. V. C., Fig. 69; see also Fig. 78). Most of these cells are fusiform in shape, but are placed vertically, so that they appear round or oval in transverse section, and their processes are not seen. Some, however, present processes that run forwards or backwards. Nerve-fibres pass into and by it, which we shall consider subsequently. Although this tract is chiefly developed in the lower dorsal and upper lumbar cord, a few nerve-cells of similar character are met with in the same position in other parts, and are occasionally sufficiently numerous to form a small group.

The "gelatinous" grey matter, *grey substance of Rolando*, which forms a cap on the posterior horn, *caput cornu posterioris* (C. C. P., Fig. 64), differs considerably from the spongy substance. It has been described as made up of granules, but, according to later researches, consists of a peculiar translucent material forming an excessively fine network, which, like the rest of the neuroglia, behaves to reagents like horny material. In the embryo it is continuous with that which surrounds the central canal,† but from this it becomes separated in the course of development. Its basis substance is thus of neuroglial nature. Numerous nerve-fibres pass through it from the posterior roots, and these, coursing forwards, seem to divide it into columns, as it is viewed in transverse section. A few small ganglion-cells lie in this gelatinous substance, chiefly on the inner side; it also contains many minute cells, some of which are thought to be nerve-cells, others to belong to the neuroglial tissue. Bundles of vertical fibres are also seen in transverse section, and these are often numerous just above the caput, especially on the inner side of the horn. Most of these are fibres of the posterior nerve-roots that have a vertical course for a short distance. In Fig. 78 the axis-cylinders of these fibres are seen to be changing from the horizontal to the vertical direction, and their relation to the posterior root fibres can be distinctly traced. The hinder part of the gelatinous substance is less dense in structure than the rest, and is sometimes distinguished from it as the "spongy zone."* The gelatinous substance contains many vessels, running for the most part vertically.

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* It is generally believed that their branching processes enter into the plexus of fibrillæ in the grey matter, and thus connect these cells with those of the anterior cornua, and also with fibres that cross by the posterior commissure and form the ascending sensory path to the brain. It has been recently maintained that there is no actual union of the fibrillæ in the grey substance, but that impulses pass from one to another by contiguity, or through a special intermediate conducting material (His and Forel, 'Cor.-bl. f. Schw. Aerz.,' 1889, Oct. 1st). This observation needs confirmation.

† Cf. Corning, 'Arch f. mikros. Anat.,' xxxi, 1658.

White Substance.—We may now examine, in greater detail, the constitution of the white columns of the cord, and the probable course and function of the fibres they contain. Their analysis has been greatly aided by three facts: continuity of degeneration, difference in time of development, and the variation in the size of the fibres of the different tracts. 1. It was discovered by Türck that in certain tracts in the white substance the nerve-fibres undergo secondary degeneration when separated from their cells of origin, a degeneration analogous to that which, as we have already seen, occurs in the nerve-fibres outside the cord. 2. In the developing cord the fibres of different tracts acquire their white substance at different periods, and the study of these differences (first by Flechsig and later by Bechterew and others) has not only confirmed, but also extended, the differential indications afforded by disease. It enables us to make a distinction between fibres that do not degenerate separately or not through a sufficiently long tract to enable us to distinguish them. The groups into which this method enables us to divide the fibres, and the order in which the medullation takes place, are as follow:—

(1) Anterior and posterior root-fibres.

$$\left.\begin{array}{l}(2)\\(3)\\(4)\end{array}\right\}\text{Ground-fibres of the}\left\{\begin{array}{l}\text{inner anterior column.}\\\text{postero-external column.}\\\text{outer anterior and lateral column.}\end{array}\right.$$

(5) Lateral limiting layer and antero-lateral ascending tract.

(6) Postero-median column.

(7) Direct cerebellar tract.

(8) Lateral and anterior pyramidal tracts (which in man are first medullated at the time of birth).†

3. The size of the nerve-fibres varies in different tracts, but the full significance of the variations has still to be ascertained. Even in the same tract of the cord considerable variations are met with, which probably correspond to differences in function. When a tract degenerates it is common for the degeneration to be incomplete; fibres remain intact among those that perish: this indicates a difference in function, and with it the difference in size usually coincides. It is also possible that some of the small fibres seen among larger ones may be due to the division of the latter (Sherrington); but such division has not been actually demonstrated, and is probably not frequent. The variation in size is from 1 to 25 μ ($\frac{1}{25000}$ to $\frac{1}{1000}$ inch). The variations are especially great in the fibres of the pyramidal tract,

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* So that in some descriptions the posterior horn is divided into (1) spongy substance of the horn itself; (2) gelatinous zone of the "caput," and (3) spongy zone at the tip of the horn. See Lissauer, 'Arch. f. Psych.,' 1886.

† See Obersteiner, loc. cit., p. 192.

and it is in these that division has been thought to occur. Those of the direct cerebellar tract are uniformly large, while most of those that constitute the postero-median column are small.

It will facilitate the detailed study of the tracts if we consider first those that undergo secondary degeneration for a considerable distance, as our knowledge regarding these is the most complete. These degenerations are both ascending and descending. Only one

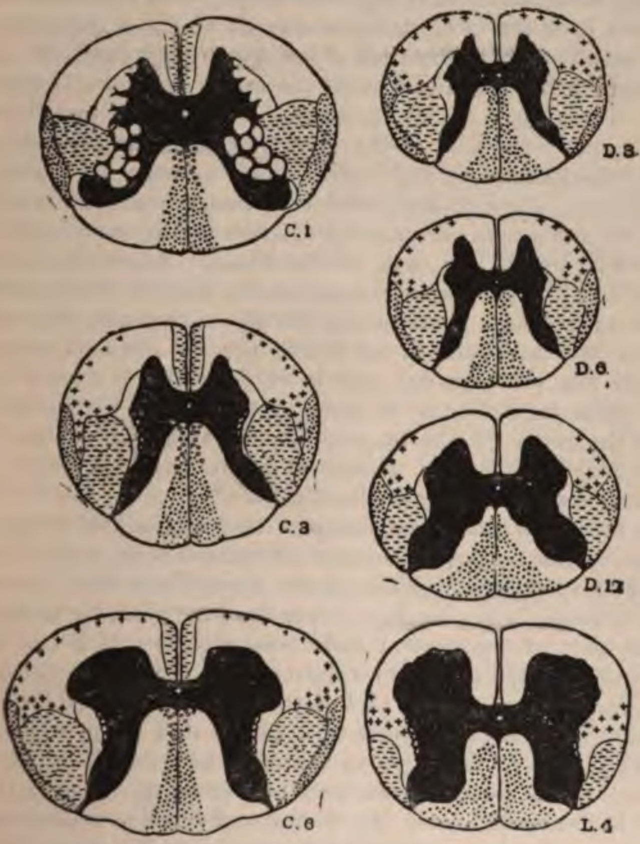

FIG. 70.—Diagram of the elements of the white substance at various levels of the spinal cord. (Modified from Flechsig.) The pyramidal tracts are shaded by short horizontal lines; the direct cerebellar tract by dots; the posterior median column by dots, as it degenerates in disease of the lowest part of the cord or of the nerves of the cauda equina; the small circles in the forepart of this column indicate the area which degenerates in disease of the cervical enlargement. The antero-lateral ascending tract is shown by crosses. It is not represented in the first cervical section because there are no facts, at present, to show its exact position here; it probably lies chiefly in front of, and between, the direct cerebellar and pyramidal tracts, as C. 3. The anterior ground-fibres, lateral limiting layer, and postero-external column are left white. Fig. 62 should be compared with this.

important set of fibres degenerates downwards through a considerable extent—those that continue through the cord the anterior pyramids of the medulla. They commence still higher, in the motor region of the cortex of the cerebral hemisphere, passing thence by the "internal capsule" between the two parts of the corpus striatum, &c., to the crus and pons; they are the chief, perhaps the sole conductors of voluntary impulses. They are termed the *pyramidal tracts* because they constitute the anterior pyramids of the medulla. They are shaded by short horizontal lines in Fig. 70; compare also Fig. 64.

At the decussation of the pyramids about three quarters of the fibres usually cross to the other side. These pass down the cord in the lateral column, and constitute the *lateral* (or *crossed*) *pyramidal tract*. Those which do not decussate, pass down their own side of the cord, in the inner part of the anterior column, at the side of the anterior median fissure, and constitute the *anterior* (or *direct*) *pyramidal tract*. Flechsig has found that the decussation is subject to many variations. In the majority of cases the decussating fibres are between 70 and 80 per cent. of the whole. Sometimes they constitute a still larger proportion, and in one case (of 60 examined) all the fibres crossed. Sometimes fewer fibres decussate, only one half or even less than half; in one case 35 per cent., in another only 10. No case has yet been met with in which no fibres crossed. A few fibres seem to decussate, at least in some cases, within the pons, where the paths for the tongue, &c., cross the middle line, since slight degeneration from a cerebral lesion has been found in the opposite pyramid of the medulla oblongata.*

The *lateral pyramidal tract* occupies the posterior half of the lateral column, outside the posterior cornu. It extends down to the end of the cord, even when it is originally small. Hence those fibres that do not cross at the medulla must do so lower down in the cord. Through the greater part of the cervical and dorsal regions this lateral tract is separated from the surface by a narrow layer of fibres, the *direct cerebellar tract*. In the upper part of the cervical region (third cervical segment) this tract lies farther forwards, so that the pyramidal tract comes up to the surface close to the posterior cornu (Fig. 68, C. 3), and here, if the tract is small, there may be a depression on the surface. In the lower part of the dorsal cord there is a similar movement forwards of the cerebellar tract, so that the pyramidal tract again comes in contact with the surface posteriorly (Fig. 68, D. 12), and, as the cerebellar tract ceases at the last dorsal nerve, the pyramidal tract extends up to the surface throughout the lumbar enlargement (Fig. 63, L 4). The inner side of the tract is in contact with the hinder part of the posterior cornu, near the surface, throughout the entire length of the cord, but further forwards it is separated from the neck of the cornu and intermediate grey substance by a layer of nerve-fibres, termed by Flechsig the *lateral limiting layer* (L. L. L., Fig. 64).

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* Hadden and Sherrington, 'Brain,' viii, 502, pl., fig. 1.

Among the fibres of the pyramidal tract, in the dorsal and cervical regions, degeneration reveals a few other fibres that belong chiefly to the cerebellar tract. The lateral pyramidal tract diminishes in size from above downwards as its fibres end by entering the grey matter; those that extend lowest (for the leg) occupy the posterior part of the tract in the cervical region. The fibres curve inwards along the septa that extend from the grey matter into the lateral column, and they enter the grey matter between the anterior and posterior cornua. They pass inwards and forwards in the anterior cornu, and are then lost in the complicated structure of the grey matter. Their probable termination will be considered presently.

The *anterior* or *direct pyramidal tract* (called also the "column of Türck") descends the cord in the inner part of the anterior column, adjacent to the anterior median fissure. Its exact form and size vary; usually it bounds the fissure on each side; sometimes it forms only a small tract at the posterior part of the fissure; when large, it not only reaches forward to the front of the cord, but may cause a prominence on the surface bounded by a distinct sulcus. Its extent down the cord, always less than that of the lateral tract, varies according to its size. It steadily lessens in area, and, if originally small, may not extend beyond the middle of the cervical enlargement; if originally large, it may be traced as far as the lumbar enlargement (Fig. 71); when bearing the usual proportion to the lateral tract, it ceases about the mid-dorsal region. Most of the fibres pass through the anterior commissure to the opposite side, either entering at once the grey matter, or passing first to the lateral tract. The latter is probably the course of most of the fibres when the pyramidal decussation is small. Whether any fibres end in the grey matter on the same side of the cord we do not know.

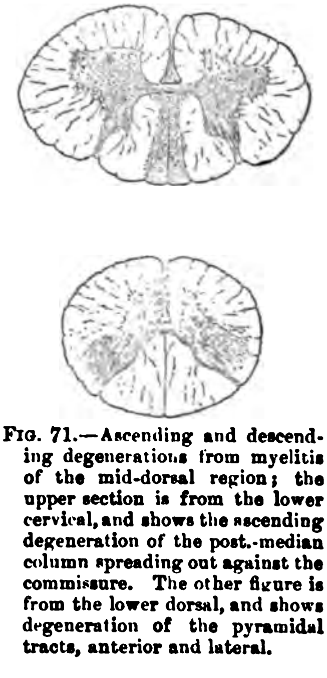

FIG. 71.—Ascending and descending degenerations from myelitis of the mid-dorsal region; the upper section is from the lower cervical, and shows the ascending degeneration of the post.-median column spreading out against the commissure. The other figure is from the lower dorsal, and shows degeneration of the pyramidal tracts, anterior and lateral.

Disease of the brain destroying the motor cortex, or the fibres which descend from it to the pyramids, causes descending degeneration, usually limited (at least in conspicuous degree) to the related pyramidal tracts,—anterior on the same side, lateral on the opposite side (Fig. 72). But in many cases slight degeneration has been also found in the lateral tract on the same side, extending into the lumbar region. Hence it is probable that some fibres of each anterior pyramid find their way to the lateral tract on the same side, and descend to the corresponding limbs, especially to the leg. This degeneration of the lateral tract on the same side as the lesion is sometimes very marked just below the decussation of the pyramids, and has therefore been thought not to be due to any lower re-decussation in the cord.* Nevertheless facts suggesting that there is such a re-decussation in dogs and monkeys have been ascertained by Sherrington,† but the anatomical relation in man between each anterior pyramid of the medulla and the anterior roots of the same side must be regarded as still uncertain. That there is such a physiological relation is clearly indicated by clinical facts. We shall afterwards see that there is abundant evidence that each hemisphere of the brain is connected with both legs, although chiefly with that of the opposite side. There is also a similar and even more equal connection with the trunk muscles, and a slighter connection with certain muscles of the arm. Such muscles invariably regain some power on the paralysed side, even with a complete interruption to the motor path, and they are weakened on the unparalysed side. The subject is considered further in vol. ii. On the whole, the facts hitherto ascertained suggest that the arrangements which subserve this relation vary in different persons, just as does the chief decussation of the pyramids. The fibres probably in some cases continue downwards on the

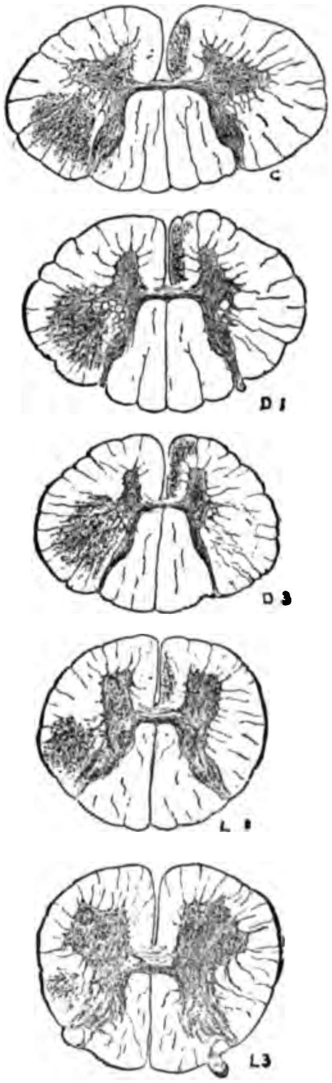

FIG. 72.—Descending degeneration of the pyramidal tracts in a case of hemiplegia from disease of the right cerebral hemisphere. (From sections prepared by Dr. Mott.)

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* Pitres, 'Arch. de Physiologie,' 1874; Hadden and Sherrington, 'Brain,' Jan., 1886. An illustration of this bilateral degeneration will be found in the section on Diseases of the Brain, vol. ii.

† "Every unilateral lesion induces degeneration in both lateral pyramidal tracts,

same side until they reach the level of grey matter for which they are destined. In other cases most or many of them cross at the bulbar decussation, to recross near the level at which they end. In this case, moreover, it is evident that the presence of visible degeneration in the lateral column, due to the decay of these fibres, will depend on the distance of their re-decussation above their ending. If they cross only when they have to enter the grey matter, they will have no recognisable place in the pyramidal tracts. Lastly, there is the possibility that a connection between the motor cells or structures of the two sides may supplement the re-decussation.*

How do the fibres of the pyramidal tract end? They seem all to pass into the intermediate grey matter, and forwards into the anterior cornu, among the motor nerve-cells. There they are lost in the plexus of the spongy substance. There is strong indirect evidence that they divide and subdivide, and that their ramifications join the plexus of nerve-fibrillæ constituted by the dividing processes of the ganglion-cells. The evidence of this is twofold. As far as is at present known, only one process of a ganglion-cell becomes an axis-cylinder; the others divide. The axis-cylinder process passes into an anterior root; it can be seen to do so in the case of some ganglion-cells (Fig. 60), and, as we have seen, the number of ganglion-cells and anterior root-fibres is nearly the same in the frog, and therefore, presumably, in man also. Since some root-fibres are not motor, the number of motor cells may safely be assumed to be at least not smaller than that of the motor fibres; thus each fibre will have at least one cell related to it. If so, since the pyramidal fibres are certainly connected with the ganglion-cells, the connection can only be by the ramifying processes of the cells, and this involves a division of the nerve-fibre. The dividing fibre isolated by Gerlach, and shown in Fig. 61, may have been a branch of a pyramidal fibre. The second point in evidence of division is the fact that the nerve-cells and root-fibres must be many times more numerous than the fibres of the pyramidal tract.† But all (or at least most) of the motor root-fibres may be stimulated through the pyramidal fibres, and therefore each of the latter must be connected with several ganglion-cells. Physiological considerations suggest the same conclusion.

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the smaller degeneration (on the same side as the lesion) being most pronounced in the cervical and lumbar enlargements, immediately above which regions it is often absent. It follows, therefore, that the degeneration on the same side as the lesion is a 'recrossed' degeneration. Further, the lower regions may even contain more degenerated fibres than the upper ones; and as degenerated fibres can often be traced for a long distance in pairs, it is inferred that the fibres of the pyramidal tracts divide into two" (Sherrington).

* Broadbent, Hadden. In a unilateral lesion of the cord the paralysed foot may move with the other. Such a connection of nuclei was originally suggested by Broadbent to explain the association of bilateral movements.

† The total area of the anterior roots of the cord is probably at least five times as great as that of the pyramidal tracts in the upper cervical region.

Let us now consider, for a moment, the whole motor path, from the cortex of the brain to the muscles. We may regard it as composed of two segments, an upper and lower (Fig. 73). Each consists of a ganglion-cell above, a nerve-fibre, and the terminal ramification of the latter. The upper, "cerebro-spinal" segment consists of the cortical ganglion-cell, and the "pyramidal" fibre, which proceeds from the cell, passes through the brain and cord, and ends in the grey substance by division and terminal connection with several nerve-cells. The lower, "spino-muscular" segment consists of the spinal motor cell, and the fibre proceeding from this, which passes through the anterior root and nerve-trunk to the muscle, where it divides and ramifies on the muscular fibre. Thus each segment has a similar constitution—cell, fibre, terminal distribution. The elements of the two segments do not correspond in number, since, as we have just seen, each cerebro-spinal element is connected with many spino-muscular elements. So, too, each motor fibre is connected with a considerable area of excitable muscle-tissue. It will be found that this conception of the motor path conduces to clearer ideas of many facts of disease, and it is important to grasp it firmly. We shall see, for instance, that diseases involving any part of a segment produce similar effects, however diverse their nature; while there is a fundamental difference between the effects of disease of the two segments.[*]

Fibres that degenerate downwards through the greater part of the cord are also scattered through the antero-lateral column, in a zone that extends beneath the surface between the direct pyramidal tract and the direct cerebellar tract. Some are mingled with the fibres of the ascending antero-lateral tract presently to be described;

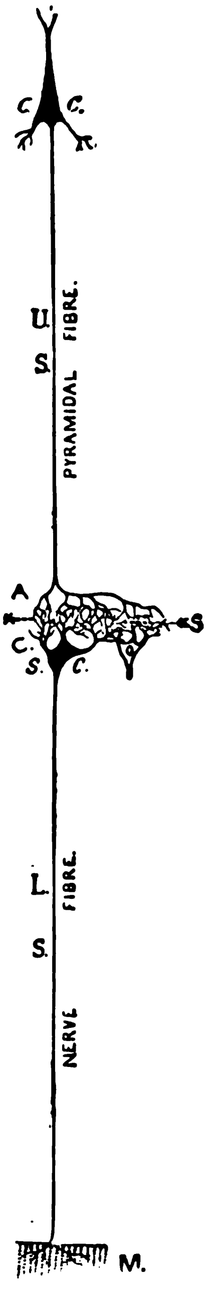

FIG. 73.—Diagram of an element of the motor path. U. S., upper segment; L. S., lower segment; C. C., cortical cell; S. C., spinal cell; A. C., anterior cornu; M., muscle; the dotted line, at the level of S., indicates the junction of the two segments; S., path from sensory nerve-roots.

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[*] I have found this conception of the motor path of so much practical value that it may be worth while to mention the curious coincidence that, a few months after its publication in the first edition of this book (in August, 1886), nearly the same

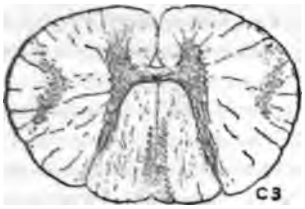

FIG. 73A.—Degeneration of the antero-lateral ascending tract in a case of locomotor ataxy (q. v. figure), showing its position in the upper cervical region, where the direct cerebellar tract passes outside it into the restiform body.

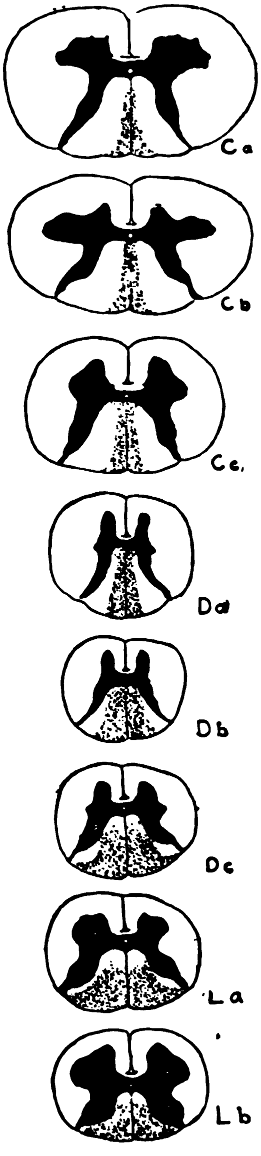

FIG. 75.—Degeneration after injury to the cauda equina. C a, b, c, upper middle and lower cervical; D a, upper; b, c, lower dorsal; L a, upper; and L b, middle lumbar region. (After Schultze.)

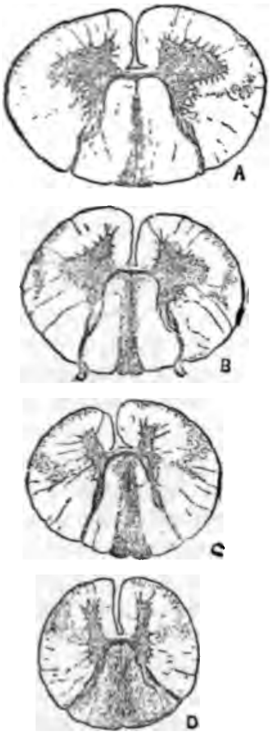

FIG. 74.—Ascending degeneration in the postero-median column and antero-lateral ascending tract. The cord was crushed at the first lumbar segment.

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dea was stated (no doubt independently), and even the same terminology employed, by Dr. v. Renz in the 'Centralblatt f. Nervenkr.,' Oct. 15th, 1886.

others lie beneath this. It is possible that they really belong to the crossed pyramidal tract. They have been termed by Foster the *descending antero-lateral tract.*[*]

Fibres degenerate upwards in both the lateral and the posterior columns. We may consider the latter first.

Each *posterior column*, as we have seen, is divided by the imperfect "intermediate septum" into two parts,—a narrow "postero-median column," and a wider "postero-external column" (Fig. 64). Secondary degeneration confirms this distinction. If the posterior columns are interrupted anywhere in their course, ascending degeneration results; and this, a short distance above the lesion, is confined to the posterior median column (Figs. 71, 74, 75), in which the degeneration continues upwards to the medulla oblongata, and ends there as the grey matter of the "post-pyramidal nucleus." The fibres of these median columns degenerate in the same way when the lesion is not in the cord, but in the posterior nerve-roots (as of the cauda equina, Fig. 70). Hence

FIG. 76.

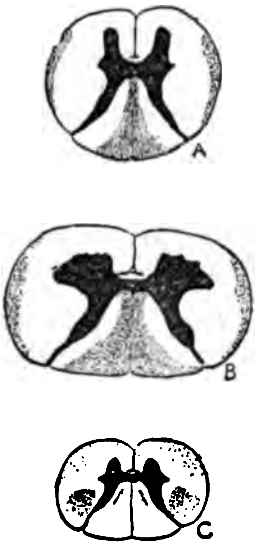

FIG. 77.

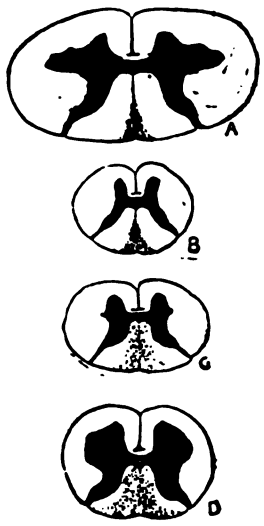

FIG. 76.—Degenerations after a lesion of the cord in the lower part of the cervical region. A, highest cervical; B, middle cervical 1½ cm. above lesion. Degeneration of the posterior median columns, spreading slightly in A and widely in B into the post. ext. col. The nature of the circumferential degeneration is uncertain; it is probably a partial ascending degeneration set up by peripheral myelitis.

C, upper dorsal 2 cm. below lesion. Degeneration of the lateral pyramidal tracts; "comma-shaped" degeneration in front of the post. ext. col.; slight degeneration of the anterior ground-fibres. (Schultze.)

FIG. 77.—Ascending degeneration after injury to the lowest part of the spinal cord and sciatic nerve-roots arising from it. A, cervical; B, lower dorsal; C, junction of dorsal and lumbar regions; D, middle of lumbar enlargement. (After Schultze.)

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[*] Foster, 'Physiology,' 1890, p. 873. See also Hadden and Sherrington, 'Brain,' vol. viii; and Tooth, 'Secondary Degeneration of the Spinal Cord,' 1889.

it is clear that these fibres are continued upwards from the posterior nerve-roots without interruption. The contrast between these degenerated columns and the undegenerated external columns through the greater part of the cord above the lesion (Fig. 74) is very striking. But just above the lesion the degeneration is not confined to the median columns; it spreads out into the external columns, especially towards the hinder surface, and the more extensively the nearer to the lesion, until close above this the degenerated fibres occupy the whole extent of the posterior column, except a small area close to the posterior cornu. This is due to the fact that the fibres pass to the median column through the hinder part of the external column. It is the same whatever is the seat of the lesion, whether it is in the cervical (Fig. 76), dorsal, or lumbar (Fig. 77) parts of the cord; and if it is in the cauda equina, the degeneration spreads out in the same way in the lumbar enlargement (Fig. 75). This shows clearly that all the way up the cord fibres pass to the median column from the external column, and that these are uncrossed fibres from the posterior roots. As the fibres ascend the cord in the median column, they come to lie posteriorly in proportion as their source is lower. Those from the sciatic nerves, for instance, in the lower cervical cord only occupy the posterior half of the columns (Fig. 77). Those from the whole lumbar enlargement, or whole cauda equina (Fig. 74), reach forward to the commissure in the lower cervical region, spreading out a little near the commissure, where, however, the degeneration is much less dense than it is behind. Above the middle of the cervical enlargement the degeneration from a lesion in the dorsal or lumbar regions does not extend forwards beyond the junction of the anterior and middle third of these columns; but if the lesion is in the cervical region the degeneration extends up to the commissure, even at the level of the second cervical segment (Figs. 76, A, 66), showing that the fibres from the cervical roots occupy the anterior portion of these columns.

It is important to remember that the root-fibres that pass to the median column through the external column do not decussate. This is not, however, the only source of fibres to the median column. Fibres pass to it (1) from the neck of the posterior horn, across the anterior part of the external column, curving backwards (Fig. 78, *x*); many of them extend almost to the postero-median septum. They come from the layer of fibres that lies in the inner part of the neck of the cornu, on the inner side of the vesicular column where this exists. Some of them may be distinctly seen to turn backwards in the neck, as if they come either from the posterior roots or from cells near the caput cornu. It is possible that others may be fibres from the posterior commissure. (2) From the posterior commissure in the middle line (Fig. 78). These are very numerous and conspicuous in the lumbar enlargement. They course backwards in the septum and then pass outwards on each side into the adjacent column.

Flechsig believes that these are posterior root-fibres. They may easily decussate in the septum, but such decussation has not been proved.

In the lower lumbar region there is a narrow tract of fibres close to the posterior median septum, that is apparently of different nature from the rest of the column. It is lenticular on transverse section, and is indicated by the clear area in L. 4, Fig. 70, and faintly in L b, Fig. 75. It is distinguished by a difference in time of development (Flechsig), and by freedom from the secondary degeneration that involves the rest of the column.

It is very doubtful whether all the fibres that enter the postero-median columns continue in these to the medulla. Some certainly do, but the upward increase in size of the columns seems to be far too small for the accommodation of all the fibres that seem to pass to them. At the same time we have, at present, no indication of the mode in which fibres leave these columns.

At the medulla oblongata this column becomes filled with nerve-cells, the *post-pyramidal nucleus*, so called because the highest portion of this column has been termed the *posterior pyramid* of the medulla. It is also called the *nucleus gracilis*. The discovery that the fibres of this column are continuous with the nerve-roots invests the post-pyramidal nucleus with considerable importance, since its

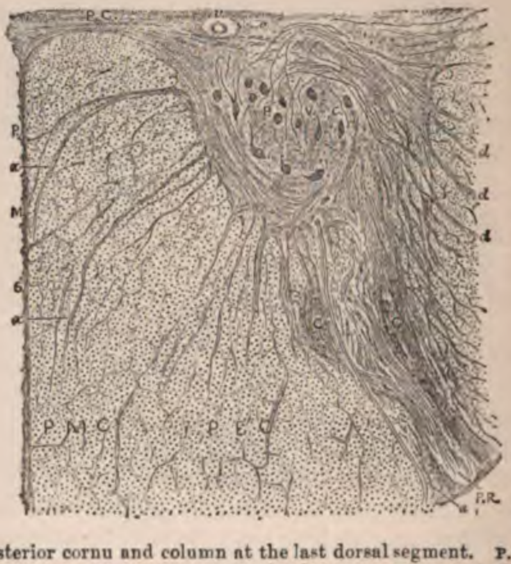

Fig. 78.—Posterior cornu and column at the last dorsal segment. P. M. C., postero-median column; P. E. C., postero-external column; P. M. S., posterior median septum; P. C., posterior commissure; *v*, commissure vein. P. V. C., posterior vesicular column; C. C., caput cornu; P. R., posterior root; *a*, an artery; *d*, *d*, *d*, adjacent to a strip of the lateral column, indicate the tracts of fibres passing from the vicinity and interior of the posterior vesicular column along the septa of the lateral column, to form the direct cerebellar tract; *x*, *x*, tracts of fibres passing from the neck of the horn, near the post. vesic. col., to the post-median column.

nerve cells are the first with which these root-fibres are connected, and they arrest secondary degeneration. The upward degeneration of the median fibres implies upward conduction; their probable function will be considered in the next section.

The *postero-external column* ("column of Burdach," containing the "posterior root-zone" of Charcot) consists chiefly of vertical fibres. Many of the posterior root-fibres pass through it, horizontally or obliquely (Fig. 64). These either curve inwards to the posterior horn (some first inclining upwards), or pass obliquely upwards and inwards to the median column and constitute the fibres of this column that are continued from the nerve-root. Across the anterior part fibres also pass, as just described, between the neck of the posterior horn and the median column. The vertical ground fibres of the external column have apparently only a short course, since they degenerate for only a few centimetres above or below a lesion. The longest descending degeneration is of a "comma-shaped tract," in the middle of the anterior third of the column in the dorsal region, which degenerates downwards for four or five centimetres (Fig. 76). These vertical fibres, of short course, may connect the grey matter of the posterior horn at different (but adjacent) levels. This column is larger in the swellings than it is in the dorsal region, chiefly in consequence of the larger number of root-fibres that pass through it. Above, it also ends in a grey nucleus, the *postero-external* nucleus or *cuneate* nucleus.

Both these posterior nuclei are connected directly with the cortex of the opposite hemisphere, and chiefly with the ascending parietal convolution. Fibres also pass from them to the cerebellar hemisphere of their own side, which is connected with the same region of the opposite cerebral cortex by the superior cerebellar peduncle.* The two nuclei (of both sides) are also found (by developmental researches) to send fibres through to the inferior cerebellar peduncle,† and fibres pass from the posterior median nucleus to the middle lobe of the cerebellum on the same side,‡ a fact of considerable significance.

The "*direct cerebellar tract*" is another series of fibres that degenerate upwards. It forms a layer at the periphery of the lateral column, outside the pyramidal tract (Fig. 64), but does not extend through the whole length of the cord, ceasing below at the level of the first lumbar nerve (Fig. 70). The anterior part of the tract (as seen in section) does not extend forwards beyond the level of the lateral pyramidal tract, although it was formerly thought to do so because there are other fibres in front of it that also degenerate upwards, and these were not distinguished from those of the tract. But near its upper and lower extremities, at the level of the second cervical nerves, and also in the lowest part of the dorsal region, the tract lies a little anterior to its position in the rest of the cord, and hence at these places the pyramidal tract comes to the surface behind the cerebellar tract, close to the posterior nerve-roots, up to which elsewhere the cerebellar tract extends. The tract increases somewhat in size from below upwards, and hence receives fibres throughout its course, but most of its constituent fibres enter it at the level of the lowest dorsal and first lumbar nerves, *i. e.* at its lowest part. These fibres come from the grey substance, passing through the lateral column, along the septa that cross the pyramidal tract from the grey matter. In the latter these fibres are conspicuous objects at this level (Fig. 78, *d*, *d*), passing transversely and obliquely from the front of the posterior vesicular column. Into this many of them can be traced; others change their direction and become vertical, perhaps passing into the vesicular column at a different level. It is probable that the fibres come from this column; the cerebellar tract is chiefly formed at that part of the cord at which the column is most developed, and the cells of this column have been found atrophied when the cerebellar tract is degenerated.* Besides the fibres that are gathered into the compact cerebellar tract, others, apparently belonging to it, ascend among those of the adjacent pyramidal tract.

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* See Flechsig and Hosel, 'Neurol. Cent.,' 1890, p. 417.

† Darkschewitsch and Freud, ib., 1886, p. 121.

‡ Bechterew, 'Wjistnik Psych., &c.,' 1886.

The cerebellar tract only degenerates when a lesion of the cord is at, or above, its place of origin, the junction of the dorsal and lumbar regions. If the lesion is in the lumbar enlargement, as in the case shown in Fig. 74, the tract does not degenerate.

At the level of the first cervical nerve (where the pyramidal tract leaves the lateral column to cross into the anterior pyramid of the medulla) the cerebellar tract lies in front of the "grey substance of Rolando" (formed from the caput cornu posterioris), and passes up, in the restiform body, to the cerebellum. Since it degenerates upwards we must conclude that it also conducts upwards.†

We have seen that the lateral pyramidal tract, although in contact, behind, with the head of the posterior cornu, does not extend quite up to the neck of the horn or to the intermediate grey substance, the two being separated by a "*lateral limiting layer*" of vertical fibres, in part broken up by processes from the grey matter. In the upper cervical cord, and also to a less extent in the dorsal cord, this layer extends forwards outside the anterior horn, which, in these parts, is narrow. It consists of fine fibres that seem to pass into and out of the grey matter. This course, and the fact that the tract does not degenerate through any considerable extent, suggest that its fibres have but a short course, and connect the grey matter of adjacent regions.

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* *E.g.* by Minkowski, 'Deut. Arch. f. kl. Med.,' Bd. xxxiv, p. 433. Slight degeneration of the pyramidal tracts was the only other lesion.

† In the absence of evidence of contrary character, the direction in which fibres conduct must be assumed to be that in which they degenerate. We know of no exceptions to it within the central nervous system. Wherever we can observe the relations this law obtains, except in the peripheral sensory nerves, in which the conditions are conspicuously exceptional. Hence the probability is very great that the correspondence obtains where we have not yet been able to prove it, and to ignore the probability because it is not a certainty is scarcely reasonable, although not uncommon.

There is a small tract of fibres, all of small size, at the junction of the tip of the posterior horn and lateral column known as Lissauer's tract. The fibres come from the posterior roots; they course upward for a short distance, forming the tract, and then enter the posterior horn.

The rest of the lateral column, in front of the pyramidal and cerebellar tracts, consists of fibres that vary in size, course, and date of development. Secondary degeneration, however, as I pointed out some years ago,* enables us to distinguish an important tract which occupies an irregular area in front of the pyramidal and cerebellar tracts, and degenerates upwards throughout the cord. It extends across the lateral column, as a band which fills up the angle between the pyramidal and cerebellar tracts, and it reaches the surface of the cord in front of the latter tract, nearly on a level with the anterior commissure; it then extends forward in the periphery of the anterior column, almost to the anterior median fissure, and up to the direct pyramidal tract when this exists. I have termed it the *antero-lateral ascending tract* (Figs. 64, 70, 73A, 74, and 79). It has often been confounded with the cerebellar tract, and hence the impression arose that the cerebellar tract extends farther forwards than it really does. Posteriorly the tract extends across the lateral column towards the posterior commissure, from which its fibres probably come (Fig. 70, L. 4). In the cervical (ib., C. 3), especially in the upper part, where the direct cerebellar tract extends farther forward, the broadest part of the ascending tract lies just within the anterior part of the cerebellar tract, and extends, like a wedge, between the latter and the pyramidal tract.

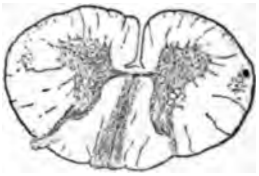

FIG. 79.—Cervical section. Ascending degeneration in post.-med. col. and ant.-lat. ascending tract, secondary to a lesion in the dorsal region. (From a section prepared by Dr. Mott.)

This tract has been found by Bechterew to undergo development at a different period from the rest of the lateral column. It apparently constitutes an upward path from the opposite posterior roots, since its degeneration, if unequal on the two sides, is greater on the side opposite to that on which the uncrossed post.-median column is most degenerated. Its fibres do not degenerate when the nerve-roots are divided, and hence probably spring from cells in which the root-fibres end. Fibres apparently pass to it through the whole length of the cord, and these are mingled together, so that the degeneration from a lesion of the lower part of the cord is not dense.* The precise form of the tract seems subject to individual variations. Above, its fibres probably end in the lateral nucleus.

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* 'Diagnosis of Diseases of the Spinal Cord,' first ed., 1879.

Some fibres of the anterior part of the lateral column are large in size, and curve upwards and downwards into the anterior horn. They must be of short course, and may be fibres of the anterior nerve-roots, which ascend and descend to nerve-cells on a different level from that of the roots by which they leave the cord.

The fibres of the anterior column, excluding the anterior pyramidal tract, are termed by Flechsig the *anterior ground-fibres* (Fig. 69). They are not separable, structurally or by development, from those of the lateral limiting layer. The ground-fibres do not degenerate through any considerable extent of the cord, and some probably connect the anterior cornua at different levels. Moreover, some of the fibres pass to the anterior commissure, and thus, by the agency of the anterior columns, a connection may be established between the two anterior cornua at different levels. Whether any of the ground-fibres are of long course but do not degenerate at all we do not know.

The *Commissure* of the spinal cord lies between the bottom of the anterior fissure and the posterior columns, and in it alone is there a passage of fibres from one half of the cord to the other. It consists of two parts, an anterior or white, and a posterior or grey commissure. The *white commissure* varies in thickness in different parts of the cord, and is largest in the lumbar region. It is composed of medullated fibres, which cross in the commissure in such a manner that the anterior fibres on each side pass out in the posterior part of the commissure on the other side. The fibres in front pass into the anterior white column, those behind into the grey substance; hence they appear to establish a connection between the anterior column of one side and the anterior cornu of the other; but many of those entering the anterior column merely pass through this to the cornu, and this is probably true also of some of the fibres that turn upward in the column. Some seem to go to ganglion-cells of the grey matter or to the fibrillary plexus, others to the anterior nerve-roots. Through this commissure must also pass the fibres of the anterior pyramidal tract, and from it may come certain fibres that have been traced across the intermediate grey substance to the lateral column, where they assume a vertical direction in the pyramidal tract, so that part of the anterior commissure is to be regarded as a continuation of the decussation of the pyramids. Other fibres may be redecussating fibres, subserving the connection of one anterior horn with the cerebral hemisphere of its own side. Lastly, a few fibres of the anterior commissure have been thought to come from the posterior nerve-roots.

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* Other illustrations of the degeneration of this tract will be found in the section on Locomotor Ataxy. For an example of its degeneration and references to previous observations, see Tooth, 'St. Bartholomew's Hospital Reports,' 1885, p. 137. See also his 'Lectures on Secondary Degeneration in the Spinal Cord,' Lond., 1890. The degeneration found by Hadden and Sherrington ('Brain,' Oct., 1888), and regarded as evidence that the tract passes to the cerebellum, is, I am convinced, the anterior extremity of the cerebellar tract. This is manifest on a comparison of fig. 5 in their plate with Fig. 79, p. 183. It is important, moreover, to note that, in many cases of transverse lesions, a superficial myelitis extending up for a short distance in the middle of each side of the cord has determined a secondary degeneration of partial, and sometimes perhaps combined character.

The *grey commissure* contains very fine medullated nerve-fibres, a large number of which pass backwards in the posterior horn of each side; some appear to be continuous with the fibres of the posterior roots, while others probably end in the nerve-cells of the posterior horn. It is highly probable that thus a considerable number of the posterior root-fibres (or fibres from cells in which the root-fibres end) cross to the other side, and that thus the decussation of the sensory path in the cord is effected, which is known to take place a short distance above the entrance of the nerves. Some of the fibres that thus cross may find their way to the antero-lateral ascending tract in the opposite lateral column, since this tract seems to begin below close to the grey matter opposite the posterior commissure. Others leave the commissure in the middle line, and pass backwards to the posterior median column, but whether these decussate or not we do not know. They are very numerous, and the lower part of the cord within the grey commissure is the *central canal* of the spinal cord, lined with epithelium, and surrounded by granular material, said to be similar in nature to that which caps the posterior horn; the two are, indeed, as we have seen (p. 169), continuous in the embryo. The canal is often filled up by epithelial débris in adult cords,* or, sometimes, is much larger than normal. The latter state probably depends on an arrest of development, for the layer of neuroglial tissue around it is much larger than usual.

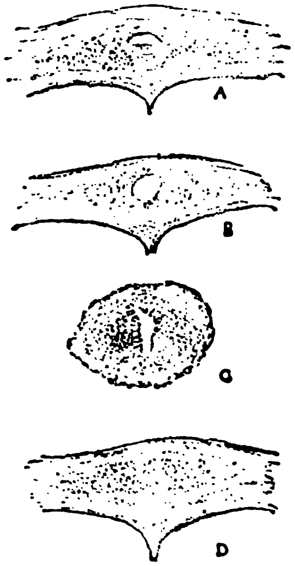

FIG. 80.—Central canal in normal cords. It has the form of a transverse slit in A, a vertical slit in C (from the conus medullaris), while in B it is circular. In each it is lined by columnar epithelium and surrounded by cellular elements, rounded, or angular from compression, mingled with granules. In D, which is from the same spinal cord as C, the position of the canal is occupied by a mass of nuclear tissue.

Root-fibres.—The course of the root-fibres has been already incidentally alluded to, but remains to be considered in a more systematic manner. The *anterior roots* pass through the anterior columns in bundles which are distributed through an area of the column approximately corresponding to the width of the anterior surface of the cornu. In the grey substance, the coarse fibres that constitute the chief part of each of these bundles pass in three different directions, outwards, inwards, and directly backwards in the middle of the horn, and thus cross each other and the fibres from the anterior commissure. Many of the fibres enter the groups of nerve-cells, and certainly end in these cells—or rather arise from them, each axis-cylinder being the prolonged chief process of the cell. Such cells often lie among the bundles outside the limit of the grey matter, and their processes can easily be traced forwards to the nerve-root and backwards into the grey matter. Some processes that pass outwards enter the lateral white column ("mixed zone"), and probably, after a short course in this, re-enter the grey matter at a higher or lower level. Some of the fibres that pass inwards may go through the commissure to the opposite anterior horn. These coarse fibres are probably all motor. Besides them, the nerve-roots contain fine fibres for the sympathetic which do not come from the cornual cells.

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* This obliteration of the canal has no pathological significance, but has been repeatedly described as one of the morbid changes in disease.

The *posterior root-fibres* have been the subject of much investigation on account of their complicated arrangement and the importance that attaches to them from their degeneration in locomotor ataxy. Although arranged in a continuous vertical series at their surface attachment, the fibres are divisible into three sets, one of which passes through the *caput cornu posterioris* (Fig. 78), a second into the adjacent part of the post-external column (Fig. 64), while a third set consists of fine fibres which at once change their direction, turning upwards, and keep together so as to form a zone (zone of Lissauer), which lies outside the extremity of the posterior horn in the cervical region, but lower down the cord between the caput and the surface of the cord.

The fibres of these three sets have only two immediate destinations —to the postero-median column and the posterior grey cornu. (1) Many of the fibres that enter the post-external column (*a*) pass obliquely upwards and inwards to the post-median column. This course is through the hinder part of the post-external column, and in the lumbar region, where the roots enter at the bottom of their furrow, many fibres actually pass backwards into the adjacent angle of the column, and may be there seen to turn upward in their ascending course to the median column. It is this arrangement that leads to the peculiar wedge-like shape of the ascending degeneration just above a lesion (Figs. 75 and 76); (*b*) other fibres entering the post-external column curve forwards and enter the posterior cornu, chiefly in front of its head. The curves they describe are largest in the lumbar region, where they almost reach the post-median column. (2) The fibres that enter the caput directly, pass through this to the posterior horn, some on their level of entrance (and these, in the lumbar region, have a curved course like the meridian lines on a globe), while others turn upwards in compact bundles to pass forward into the cornu at a higher level. (3) The fibres of Lissauer's group also enter the horn, either through or in front of the caput, after a short ascending course.

Thus, except the fibres to the post-median column (which ascend in it without decussating), all the other posterior root-fibres enter the posterior horn, chiefly on its inner side or through the caput, a few (Lissauer's) on its outer side.*

In the posterior horn, fibres of each set pass to the posterior commissure; others go to the posterior vesicular column (where this exists), while some pass forwards into the anterior cornu, chiefly towards the outer group of nerve-cells; a few pass towards the anterior commissure, and a considerable number probably end in the nerve-cells of the horn. It is probable that the fibres that pass to the posterior vesicular column† are connected with its cells, most likely indirectly through their ramifying processes and the spongy plexus, because the fibres of the plexus have been found atrophied with those of the posterior root. Many of those that pass towards the posterior commissure are probably also interrupted by nerve-cells in the posterior cornu. Thus the chief destination of the fibres that do not enter the postero-median column must be regarded as the anterior cornu of the same side and the opposite half of the cord; and this is probably true also of the numerous fibres that end in the nerve-cells of the cornu. The fibres from the vesicular tract to the direct cerebellar tract perhaps continue the path of the posterior root-fibres.

BLOOD-SUPPLY TO THE SPINAL CORD.—The arrangement of the arteries in the cord is not a matter, at present, of much practical importance. Nevertheless it is one on which some detailed facts are necessary, because it is a subject to which it is most desirable close attention should be given, on account of the probability that vascular lesions are more frequent than our present knowledge indicates, and the relation of lesions to arterial regions should be carefully noted. The chief general fact is that the blood-supply is in part central, in part peripheral: the central supply is derived from the branches in the anterior median fissure and embraces the grey matter, except the posterior horn; the peripheral supplies the posterior horn (except the neck) and the white substance.

The arterial blood is brought to the cord by small branches, derived from the vertebrals, intercostals, and other arteries, which reach the cord by the anterior and posterior roots. The anterior pass for the most part inwards, to the anterior median fissure, where they are connected by vertical branches, continuous in direction, so as to form an *anterior spinal artery.* From this a series of branches pass backwards in the anterior median fissure, which may be called *anterior median arteries,* and are of great importance, supplying most of the grey matter. At the bottom of the fissure, each divides into two branches, a right and left *commissural artery,* which passes outwards and backwards through the commissure, displacing its fibres (and hence, in section, the commissure often appears to be interrupted where the fibres are divided obliquely). At the end of the commissure each divides into an *anterior central artery,* which supplies most of the anterior horn, and a *posterior central,* which supplies the intermediate grey matter and the neck of the posterior horn, including the region of the posterior vesicular column. Each commissural artery, moreover, before dividing, gives off a branch which immediately bifurcates into an upward and downward vessel, each continuous with a corresponding branch from the next commissural artery above and below — the *anastomotic artery.* This effects a vertical continuity of anastomoses within the cord, like that of the anterior spinal artery outside the cord.

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* The fibres that pass to the post-external column have been termed the "median group," and the rest of the root-fibres the "lateral group," those entering the caput being distinguished as an "intermediate group." But each investigator describes much the same facts in a different method, and adopts an original nomenclature, which renders it better at present to keep the facts more prominent in the mind than the names.

† According to Mott the fibres divide and form a fine plexus before joining the cells of the vesicular column.

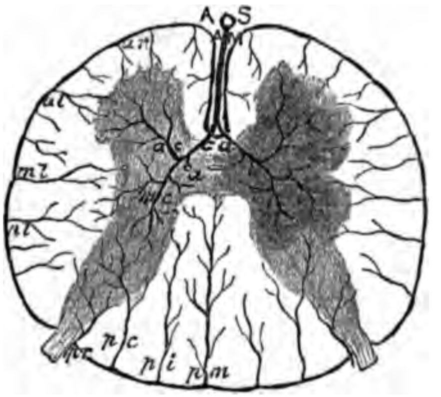

FIG. 81.—Semi-diagrammatic representation of the arteries of the spinal cord; A S, anterior spinal. *Central arteries.*—A M, anterior medial; *c a,* between the right and left commissural arteries; *a,* anastomotic artery, divided transversely, to which a branch goes from the commissural artery, which then divides into *a c,* anterior cornual, and *m c,* mid-cornual arteries. *Peripheral arteries.*—*p m,* posterior medial; *p i,* post-intermediate; *p c,* posterior cornual; *p r,* posterior radicular; *p l, m l, a l,* regions of the posterior, middle, and anterior lateral branches; *a r,* anterior radicular. In the right half of the figure the more deeply shaded part indicates the region supplied by the central arteries.

The *peripheral arteries* pass inwards from the surface. A *posterior medial* artery courses in the median septum, giving branches to each side, and an *intermediate septal artery* passes in the corresponding septum outside the postmedian column. Midway between this and the root-furrow, a branch enters and passes through the post-external column to the posterior horn, which it enters in front of the caput and chiefly supplies—the *posterior cornual artery;* while a *posterior radicular artery* passes in on the inner side of the posterior root, and is distributed to this and the caput. A series of *anterior radicular* arteries enter with the anterior roots, and between them and the posterior horn are *anterior*, *middle*, and *posterior lateral* arteries in corresponding parts of the lateral column. The branches into the lateral column do not reach the grey substance; the branches in the grey substance extend into the inner part of the lateral column.

The *veins* of the cord correspond in the main to the arteries, but a larger proportion of the blood passes into the peripheral system. Some, especially those of the white substance, pass outwards along the septa, and those on the surface of the sides and back of the cord are gathered into a *posterior spinal vein*. Others, especially of the grey matter, pass inwards to a large vertical vein that lies in the grey commissure, a little distance from the central canal on each side (*v.* Fig. 73; compare also Fig. 57), from which branches pass forwards to the anterior median fissure, and through this to an *anterior spinal vein*. The anterior and posterior spinal veins deliver their blood, through communicating veins, into the large venous plexuses that lie outside the dura mater, and which receive blood also from the bones, and from the structures and skin behind the spine. But the veins of the spinal cord cannot be injected from these plexuses, not because there are valves in the connecting branches, but apparently because they form so trifling a proportion of the total connections. From the plexuses blood passes to the various vertebral, cervical, and intercostal veins.

Although there is a vertical continuity of the vessels of the cord, it is probable that the course of the circulation is, in the main, horizontal. From the very tortuous course of the path by which arterial blood enters the cord, it is evident that the pressure to which the arteries of the cord are exposed must be far lower than that in the arteries of the brain, and hence they are far less liable to degeneration and to rupture. On the other hand, the conditions that prevent an injection of the plexus of veins outside the dura mater from passing into the veins of the cord, must save the latter from the extreme over-distension to which they would otherwise be liable when there is a hindrance to the return of blood from the plexus.

FUNCTIONS OF THE SPINAL CORD.

We are now in a position to consider the functions of the spinal cord, and the structures by which they are subserved. The functions are these: (1) The spinal cord conducts motor nerve impulses from the brain and sensory impulses to it. (2) It constitutes a series of centres for reflex action. (3) It contains certain centres that ultimately govern nervous action in structures under the immediate control of the sympathetic system, the bladder, rectum, blood-vessels, &c. (4) It influences the nutrition in all parts to which its nerves extend.

MOTOR CONDUCTION.—The chief facts regarding the physiology of the motor path have been necessarily considered in the account of its anatomy. We have already seen that the path of motor impulses is through the pyramidal fibres (in the anterior and lateral pyramidal tracts), the anterior cornua, and the anterior nerve-roots. The ganglion-cells form part of the path, and so also probably does that part of the plexus of fibrillæ in the cornu which intervenes between the pyramidal fibres and the ganglion-cells. We have seen that the crossed relation of the cerebral hemisphere with the limbs is subserved by the decussation—chiefly at the pyramids, partly along the cord from the one anterior to the opposite lateral tract, and apparently to a slight extent above the chief crossing, since some degenerated fibres have been seen in the anterior pyramid opposite to the lesion of the brain. We have seen also that we are still ignorant of the mode of connection of the motor cells with the cerebral hemisphere of the same side in the case of the muscles that habitually act with their fellows on the opposite side. These can be acted on by either hemisphere in degree that approaches equality in proportion as they act together, and is almost complete in muscles such as the intercostals, which can only act together. It is great in the case of all the trunk muscles and of those of the leg, less in those of the arm. This relation is further considered in the account of hemiplegia in vol. ii, to which it specially pertains. The variations in the decussation of the chief set of fibres suggest that the variations in observed facts bearing on the question may merely represent variations that exist in nature. The connection may be subserved by fibres that never decussate, and by fibres that recross in the cord, in different degrees in different persons.

The large fibres of the anterior, motor, nerve-roots are far more numerous than the fibres of the pyramidal tracts, and so also are the cells of the anterior horn, from which the large root-fibres proceed. Nevertheless, it is probable that all these cells and root-fibres can be excited through these tracts. Hence many cells must be related to each of the pyramidal fibres. We do not know precisely how the latter end, but it is probably a complex mechanism, constituted by minute nerve-cells and fibrillary network. This mechanism is such that the stimulation of certain pyramidal fibres excites to action a much greater number of nerve-cells, so connected and arranged as to produce, through the related nerve-roots, a complex movement, in which many muscles may take part. The nerve-cells thus connected may not all be at precisely the same level, and only some of those at a given level may be thus associated. The simpler the movements, and the fewer their possible variations, the fewer pyramidal fibres may suffice for the production of the movements. Thus it is conceivable that for such movements as those of the intercostal muscles, which are simple and constant, very few pyramidal fibres may suffice, whereas a much larger number must be necessary for the highly variable movements of the hand. It is also possible that the pyramidal impulses pass not only through their own end-structures, but also through part at least of the muscle-reflex centre, and that this enters into the mechanism that enables these impulses to effect a more complex and varying movement than could otherwise be produced.

Representation of Movements in the Spinal Cord.—It is important to inquire how far we can identify these various motor mechanisms of the anterior grey matter. There are several sources of information on this subject. We have already seen that the arrangement of fibres in the anterior roots is such as to associate certain movements with certain nerve-roots, and it is a reasonable assumption that this corresponds, to some extent at least, with the arrangement in the grey matter. The sources of our information on this point are the limited facts of anatomy, rare cases of restricted disease of the nerve-roots in man, and especially the experiments of Ferrier and Yeo on monkeys. We have already considered the indications that these experiments afford, and their suggestiveness, and we have seen that their value is relative rather than absolute. As regards the grey matter, we must remember also that there may not be a strict horizontal correspondence between the nerve-roots and the nerve-cells, because it is probable that some root-fibres enter the antero-lateral white columns, and are connected with nerve-cells at a higher or lower level than that at which they leave the cord. Another source of information is the degeneration of nerve-cells that follows slowly on an amputation of a limb, and the condition of the spinal cord in congenital absence of part of a limb. The last and most important source of information is supplied by cases of limited disease of the anterior cornua, in which the position of the lesion and the distribution of the resulting palsy can be compared. Destruction of nerve-cells causes degeneration of the motor fibres proceeding from them, and wasting of the muscles to which those motor fibres proceed. This alone affords us certain knowledge. Cases of clear significance are, however, rare, and it will be long before our knowledge can be complete. Meanwhile, we may learn something of the central association of muscles by observing what muscles are most frequently paralysed together by such disease. This subject has been carefully studied by E. Remak,[*] and by Thorburn,[†] and many valuable isolated observations have been published. It is only the associations which are frequent that can be allowed significance, because it is not uncommon to have more than one focus of disease in the grey matter. The following summary presents the conclusions that seem probable from the evidence at present available.[*]

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[*] 'Archiv f. Psychiatrie,' vol. ix.

[†] 'Brain,' January 7th, 1887, and October, 1888. The facts were observed on cases of injury. He associates each muscle with a single spinal nerve, but these nerves are only collections of roots that are severally continuous, as are the groups of nerve-cells within the cord, and it is certain that the roots related to many of the muscles go into adjacent spinal nerves.

The various facts for the most part fully confirm the conclusion of Remak, corroborated by the experiments of Ferrier and Yeo, that most movements and muscles are represented in vertical tracts, and the whole anterior grey matter, at any one nerve-segment, contains cells that are concerned with different movements. An extensive lesion of small vertical extent may thus weaken many movements, but abolish none. The special representation of the muscular functions, *i. e.* of definite movements, is no doubt related to special groups of nerve-cells, but a single group may be concerned in more than one associated movement. Different groups are probably intimately connected, because we know that the contraction of any muscle is accompanied by a slighter but proportioned contraction of its antagonists. Anatomical connections in both spinal cord and brain doubtless underlie this association.

CERVICAL REGION.—The highest cervical region apparently contains centres for the small rotators of the head, and the depressors of the hyoid bone were found by Beevor and Horsley to be related to the first two pairs of nerves, the sterno-hyoid and sterno-thyroid chiefly to the first and the omohyoid to the second.[†] The upper part also contains centres for the muscles of the neck, especially for the sterno-mastoid and upper part of the trapezius. The diaphragm is probably represented in the grey matter at the level of the roots of the phrenic nerve, the fourth cervical. In the brachial region of the cervical enlargement, the muscles of the shoulder are represented chiefly in the upper part; the intrinsic muscles of the hand in the lower part. The flexors of the elbow are represented above its extensors, and the supinators and extensors of the wrist above the flexors of the wrist.

Deltoid, scapular muscles, pectoralis, and *serratus.*—The fifth and sixth segments, for the most part the sixth, and probably the outer group of nerve-cells.[‡] The centres for the two parts of the pectoralis are separate, and are associated—the clavicular with that of the serratus magnus, the costal with that of the latissimus dorsi (see p. 30). These associated muscles are represented near together, but not at the same spot; the association is often reproduced in disease of the anterior cornu, the clavicular part being affected together with the serratus, but one muscle may suffer without that which is commonly associated with it.

The *Flexors of the elbow* and *supinators* probably correspond nearly in level with the deltoid. The whole of this series of muscles may be affected alone by disease of the nerve-roots (see p. 91) or of the grey matter, and then we have the "upper arm type" of palsy, described first by Erb.

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[*] A useful summary of the evidence up to the middle of 1884 was given by Dr. Allen Starr, in the 'American Journal of Neurology,' Aug. and Nov., 1884. The subject is a tempting one for theory, but it is very important to keep to the solid ground of facts, however limited it may be in extent.

[†] Chiefly the posterior belly of the muscle, by a branch joining the glosso-pharyngeal. The other muscles named (including slightly the anterior belly of the omohyoid) are innervated from the first cervical by a branch joining the hypoglossal.

[‡] Thorburn (loc. cit.) refers the infra- and supra-spinata with much probability to the fourth pair of nerves.

Extensor of the elbow (*triceps*): probably the middle of the brachial region, chiefly the seventh segment, extending down to the eighth, and perhaps up to the sixth, possibly chiefly in the outer group of nerve-cells.

The *Extensors of the wrist* are represented above those of the elbow; probably chiefly at the sixth or sixth and seventh segments, and not from the postero-external, but from the other groups. *Flexors of the wrist:* below the extensors, nearly on the same level as the extensors of the elbow, at the seventh and eighth segments, and probably also from one of the outer groups of cells. The *pronators* have nearly the same representation as the flexors of the wrist.

Long Extensors of the fingers: upper part of the brachial region, about the level of the sixth or between it and the seventh segment; probably from one of the anterior groups (not from the postero-lateral group). *Long Flexors of the fingers:* below the extensors; probably seventh or eighth segment.

Intrinsic muscles of the hand: lowest part of the brachial region, and the thenar muscles a little higher than the interossei; the latter being represented in the first dorsal segment.[*] The intrinsic muscles (like the long extensors) are probably related less to the outer than to the inner and anterior groups. The two sets of muscles are related both in action and in central connection, as was illustrated by a case of concussion-lesion under my care, in which the two outer interossei were paralysed and wasted, and also the part of the long extensor supplying the two outer fingers, but no other muscles.

LUMBAR ENLARGEMENT.—Our knowledge of the representation of muscles in the lumbar enlargement is very slight, and we have scarcely any definite facts concerning their relation to the cell-groups. The postero-external group preponderates over the others in size, even more than in the cervical cord, and doubtless also in importance. The few pathological observations of changes in the several groups are not altogether consistent. The chief facts available relate to the probable segmental level and association of the centres, and are as follows:

Cremaster, second lumbar segment. *Psoas*, second, *iliacus*, third, lumbar; the two muscles (one in function) are probably related to a group of cells extending through both segments. *Adductors*, fourth lumbar segment. *Gluteal muscles* (extensors of hip), fourth and fifth lumbar segments.

Extensors of knee, third and fourth lumbar segments, probably from the same group as the flexors of the hip; the two sets of muscles are often affected together, and sometimes the adductors suffer with them. The *Sartorius* is probably related to the third segment, but not to the same cell-group as the extensors of the knee (to which, indeed, it does not belong; see p. 40). It usually escapes in atrophic palsy of the extensors. *Flexors of knee*, fifth lumbar and first sacral segments. Their centre is thus below and certainly distinct from that of the extensors.

The muscles of the lower leg, moving the foot and toes, are related to the

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[*] This is the indication of the majority of cases. Nevertheless Sahli has recorded a case of atrophy of all the intrinsic muscles of the hand, in which the lesion did not extend below the seventh segment: the eighth and first dorsal were perfectly normal. I have seen a case of atrophy limited to the deltoid and intrinsic muscles of the hand, due to an acute process, probably hæmorrhage, but in this case there may have been two lesions.

fourth and fifth lumbar and the first sacral segments. The *calf muscles*, the *tibialis anticus*, and the *peroneus longus* have certainly separate centres, and are often affected separately. The tibialis anticus may suffer alone, or may alone escape when the other muscles in front of the leg are paralysed. The muscles of the calf may be affected when those in front of the leg are not. It is probable that the peroneus longus is the highest in central representation, and is related to the fourth lumbar segment.

The *intrinsic muscles of the foot*, especially the interossei, are the lowest in central relations, being connected with the second sacral segment. The position of their centres, in the lowest part of the lumbar enlargement, thus corresponds to that of the analogous muscles of the hand, in the lowest part of the cervical enlargement.

Sensory Conduction.—Our knowledge of the sensory path in the cord is far less definite than that of the motor path, in spite of the fact that it has been the subject of a large number of experimental investigations. The subject is a very difficult one for experimental solution, on account of the difficulty of ascertaining the condition of sensation in animals. The indications afforded by disease are equivocal for another reason. We have seen (p. 60) that, in the sensory nerves, a lesion which permanently interrupts motor conduction may scarcely affect sensory conduction, and the same thing is apparently true of the spinal cord. If loss of feeling results from an acute lesion, it often quickly passes away, although motor palsy remains complete and absolute, and this when all the elements of the cord seem equally implicated in the morbid process. In cases of such chronic disease as compression, which when slow must act on all the structures in nearly equal degree, the same escape of sensory conduction is often observed. Difficulties are encountered whatever mode of interpretation of the phenomena we adopt, and the fact of importance to our present problem is that we are not yet able strictly to compare structural and functional damage when the former is moderate. When the damage has been slowly produced it may be apparently great, and yet function may not be much impaired, especially the lower function of conduction of nerve-force. The significance of these facts as regards the problem of sensory conduction in the cord is that we must be cautious in drawing any conclusion from the persistence of sensation. We cannot infer, because supposed sensory tracts are visibly diseased in a case in which there was no loss of sensibility, that these tracts have not the function assigned to them, unless we can feel sure that all the axis-cylinders are destroyed. This we can seldom do; and for this reason, and because morbid processes are often wide in distribution, the evidence that can be obtained by comparing the position of lesions with the sensory symptoms they produce is limited and uncertain. Other sources of information are the effect of partial lesions of the cord, intense in degree, but limited in area, especially traumatic lesions affecting one half of the cord or part of one half. Cases of this kind are of high value, but in few, at present, have the extent of lesion and the range of symptoms been accurately ascertained.

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But how few fibres there are, in either situation, that can subserve the function. The fibres of the posterior median column pass up the same side of the cord, and in the posterior columns elsewhere we have, besides the remaining root-fibres and the "comma tract," an apparently insignificant number of fibres, and these do not degenerate through any considerable extent of the cord. Nevertheless the front of the post.-external column contains a very large number of exceedingly small nerve-fibres, which have attracted less notice than they deserve. In the lateral column, moreover, we must, in like manner, dismiss the pyramidal and cerebellar tracts—the latter because disease has proved to us that the path of these sensations is by the pons and not the cerebellum. Still, we have here the antero-lateral ascending tract, and we have also the ground bundles, and the question presents itself which seems to have been tacitly taken as a greater difficulty than it really is—does the absence of the continuity which would cause continuous degeneration, exclude such conduction as we need? The fibres that do not degenerate through long courses may degenerate through short tracts, passing into the grey matter to nutrient cells. Prof. Foster has suggested such an "in and out" path as probable for this sensory conduction. But there are many difficulties in the conception of such very frequent interruption by nerve-cells as this theory involves. A special obstacle, moreover, to the opinion that any part of the path involves the grey matter is the invariable escape of sensation in anterior polio-myelitis, which must often involve at least the intermediate grey substance. But it is possible that there may be many fibres which undergo no secondary degeneration, and yet habitually conduct in one direction. Because we admit, as we probably ought to admit, that degeneration in a given direction means habitual conduction in that direction, it does not follow that the absence of degeneration means the absence of such habitual conduction, although the assumption seems to underlie a good deal of current reasoning on the subject. There are many fibres of unknown function, which do not degenerate, scattered even among those of the posterior median column, and there are still more in the post.-external column, while they are comparatively abundant in the anterior column.

Still, they are not numerous enough to save us from the second difficulty, that the fibres we can look to are, taken altogether, insufficient in number to subserve the marvellous differentiation of sensation in the periphery—differentiation in quality of feeling and especially in locality of feeling. But a difficulty may become inoperative, without ceasing to exist, by being absorbed in a still larger difficulty of the same kind. If we avail ourselves of all the structures of the spinal cord that can conceivably subserve upward conduction, we fail to find room for the separate conduction of impulses which, nevertheless, the spinal cord must separately convey.

On only one hypothesis is it possible to conceive such conduction. The increased power of the microscope has shown us that each axis-cylinder, so called, is apparently not a simple body, but a compound structure, composed of fibrillæ, and comparable, in point of fact, to a bundle of nerve-fibres. The sensory axis-cylinders divide at their peripheral distribution in the skin, and we can scarcely conceive them dividing except by their fibrillæ. It seems to follow of necessity that each sensory area of distinct perception must be subserved by one of these fibrillæ. It is conceivable that the impulses may keep each to its own fibrillary path, even if an axis-cylinder contain as many fibrillæ as a nerve does fibres. We have, indeed, seen that such fibrillæ seem to keep separate even in traversing a nerve-cell. It is only by such an arrangement that it seems possible for the sensory impressions from skin and tissue, of touch, pain, and temperature, to be carried to the brain so as separately to excite certain nerve-cells of the cortex.

If these considerations have weight we cannot exclude the lateral and posterior columns from the function of sensory conduction on account of the scanty fibres we can discern to subserve the function, and may consider what evidence on the point disease and injury afford in man.

Unilateral lesions of the spinal cord, which cause loss of sensibility of the skin, do so on the side opposite to the lesion. This is true of all forms of sensibility. They thus prove, beyond doubt, the decussation in the spinal cord of the path from the cutaneous nerves of pain (*i. e.* common sensibility), of touch, and of temperature. An analysis of the cases in which both effect and lesion have been exactly ascertained —which will be considered more fully when these lesions are described —suggests certain other conclusions regarding the position of these paths. The facts suggest, first, that the paths for sensations of pain and of touch are not near together. Sensibility to pain has been lost in almost all recorded cases, but that to touch in only two thirds. They suggest, secondly, that the two paths for tactile sensibility are nearer together than are the two paths for common sensibility. In no case of chiefly unilateral lesion has sensibility to pain been lost on both sides, whereas in two recorded cases sensibility to pain was lost on the side opposite to the lesion, while that to touch was lost on both sides.[*] Both these conclusions harmonise with the suggestion of experiment, that painful sensations are conducted in the lateral column, those of touch in the posterior column. These conclusions, moreover, are supported by the facts of two important cases of injury to the cord which afford direct localising indications. One is a case, recorded by myself,[†] in which the spinal cord in the upper cervical region was damaged by a small spiculum of bone being driven against it by a bullet, which, entering the mouth, lodged in the body of the third cervical vertebra. The extent of the lesion of the cord is shown in Fig. 82. The chief injury is clearly to the lateral column and grey matter, the posterior column being merely swollen, apparently by œdema. The affected part was the seat of hæmorrhage and hæmorrhagic infiltration. The effect was entire loss of sensibility to pain on the opposite side without any impairment of tactile sensibility. To this the other case, recorded by Müller,[‡] is almost a complement. A stab-wound divided the whole of one half of the cord, including the posterior column, and also the posterior column of the

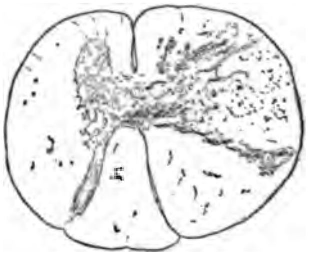

FIG. 82.—Section of spinal cord between the second and third cervical nerves, contused by gunshot injury.

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[*] It is possible that tactile sensibility may have been lost, in other cases, on the second side, *i. e.* on the side of the lesion, and the loss may have escaped notice in consequence of the exaltation of sensibility which is commonly present, and which is presumably due to the nerves of common sensibility.

[†] 'Clinical Society's Trans.,' vol. xi, 1878, p. 24.

[‡] 'Beiträge zur Path. Anat. und Phys. der Ruckenm.,' Leipzig, 1871. Abstracts, which supplement each other, and, together, give a full account of the case, will be found in Köbner's article on unilateral lesions, 'Deut. Arch. f. klin. Med.,' 1877, Bd. xix, p. 190; and in 'Virchow's Jahresbericht,' 1871, Bd. i, p. 152.

allowed significance, because it is not uncommon to have more than one focus of disease in the grey matter. The following summary presents the conclusions that seem probable from the evidence at present available.*

The various facts for the most part fully confirm the conclusion of Remak, corroborated by the experiments of Ferrier and Yeo, that most movements and muscles are represented in vertical tracts, and the whole anterior grey matter, at any one nerve-segment, contains cells that are concerned with different movements. An extensive lesion of small vertical extent may thus weaken many movements, but abolish none. The special representation of the muscular functions, i. e. of definite movements, is no doubt related to special groups of nerve-cells, but a single group may be concerned in more than one associated movement. Different groups are probably intimately connected, because we know that the contraction of any muscle is accompanied by a slighter but proportioned contraction of its antagonists. Anatomical connections in both spinal cord and brain doubtless underlie this association.

Cervical Region.—The highest cervical region apparently contains centres for the small rotators of the head, and the depressors of the hyoid bone were found by Beevor and Horsley to be related to the first two pairs of nerves, the sterno-hyoid and sterno-thyroid chiefly to the first and the omohyoid to the second.† The upper part also contains centres for the muscles of the neck, especially for the sterno-mastoid and upper part of the trapezius. The diaphragm is probably represented in the grey matter at the level of the roots of the phrenic nerve, the fourth cervical. In the brachial region of the cervical enlargement, the muscles of the shoulder are represented chiefly in the upper part; the intrinsic muscles of the hand in the lower part. The flexors of the elbow are represented above its extensors, and the supinators and extensors of the wrist above the flexors of the wrist.

Deltoid, scapular muscles, pectoralis, and *serratus.*—The fifth and sixth segments, for the most part the sixth, and probably the outer group of nerve-cells.‡ The centres for the two parts of the pectoralis are separate, and are associated—the clavicular with that of the serratus magnus, the costal with that of the latissimus dorsi (see p. 30). These associated muscles are represented near together, but not at the same spot; the association is often reproduced in disease of the anterior cornu, the clavicular part being affected together with the serratus, but one muscle may suffer without that which is commonly associated with it.

The *Flexors of the elbow* and *supinators* probably correspond nearly in level proof that these root-fibres pass up without interruption, the muscle-reflex action must be subserved by other fibres. For it, however, comparatively few may suffice. With regard to the other sensory fibres, the evidence at present suggests that all undergo interruption in the spinal cord, since no other paths have yet been found degenerated when a lesion has been confined to the nerve-roots. If so, the same fibres probably subserve sensation and reflex action, the nerve-cells or fibrillary plexus providing a mechanism for the division of the path and diversion of the impulses. No impression, in health, causes a true reflex action that does not also produce a conscious sensation.

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* A useful summary of the evidence up to the middle of 1884 was given by Dr. Allen Starr, in the 'American Journal of Neurology,' Aug. and Nov., 1884. The subject is a tempting one for theory, but it is very important to keep to the solid ground of facts, however limited it may be in extent.

† Chiefly the posterior belly of the muscle, by a branch joining the glosso-pharyngeal. The other muscles named (including slightly the anterior belly of the omohyoid) are innervated from the first cervical by a branch joining the hypoglossal.

‡ Thorburn (loc. cit.) refers the infra- and supra-spinata with much probability to the fourth pair of nerves.

We know very little of the serial representation of cutaneous sensibility in the spinal cord. It is probable, however, that there is a progressive representation of the skin in the posterior nerve-cells without relation to the nerve-trunks and with only a slight relation to the nerve-roots. Correspondence with the latter must not be assumed. In the roots the serial representation is along the axis of the limb, not across it, as it probably is in the nerve-cells. In the dorsal cord, ascending disease often causes an affection of sensation which extends around the trunk at higher and higher levels. In the limbs we have indications of the same arrangement: the soles and palms may be affected alone, and anæsthesia may extend on the limb to a certain level, irrespective of nerve distribution. This is often seen in the legs. I have known anæsthesia in the arms, due to disease of the cord, to cease midway between shoulder and elbow, at the same level around each arm. Disease of the cord may affect sensation on the tips of the fingers only. It is probable that the ascending course of many of the root-fibres subserves a rearrangement of representation in the cells, and provides also for a special connection of the tactile nerves from each part of the skin with the motor cells for the muscles beneath —so conspicuous in many of the cutaneous reflexes.

Reflex Action.—The grey matter of the spinal cord constitutes a series of reflex centres, some of which must be of considerable vertical extent and much complexity, and are subserved possibly not only by the grey matter, but by the short fibres that in the several white columns seem to connect the grey matter at different levels. The passage of the root-fibres upwards and downwards in the cord, before they turn into the grey matter (conspicuous in the posterior roots), also, as we have noted, constitutes an arrangement for spreading the afferent impulses through a considerable vertical extent of the cord.

We have already seen (p. 14) that we must distinguish two forms of reflex action—the cutaneous reflex action and the muscle-reflex action, the latter producing the "myotatic irritability" which, as we assume, determines the so-called "tendon-reflex contractions." The first form of reflex action is not, however, limited to impressions on the cutaneous nerves. It may be produced by stimulation of the nerves supplying the deeper structures, including those of the tendons and muscles. The attempt to obtain the knee-jerk may cause a true reflex action as well as its special effect, the two being separated by an appreciable interval of time (see "Locomotor Ataxy"). Reflex action from the skin is apparently subserved by all the sensory nerves, since it may continue, although the nerves for either pain or touch have ceased to act, in consequence of those degenerative processes by which function is sometimes abolished in as complete degree and with as precise limitation as the physiological inquirer could desire.

The central process concerned in reflex action must take place between the posterior nerve-roots, by which the sensory impulses reach the cord, and the anterior roots by which the motor impulses leave it. The motor ganglion-cells form part of the reflex centre, and the rest of the centre is doubtless constituted by part of the fibrillary plexus, the minute nerve-cells within it, and also by the sensory nerve-cells. The whole path between and including the in- and out-bearing nerves may be spoken of as the "reflex arc;" the grey matter concerned being called the "reflex centre." In this "centre" the sensory impulse is transformed into—or rather gives rise to—a motor impulse, usually much greater in the degree of its energy. Within the centre there are paths established by the connections between the nerve-structures, of which only a few are in functional use. It is easy to conceive that the functional action takes place more readily in some lines than in others, and these lines of easiest action are often spoken of as "lines of least resistance," and are said to determine the form of the reflex action.

The stronger (within limits) the afferent stimulus, the more widely does the process spread in the centre, and the more extensive is the movement produced. Thus a slight touch on the sole may cause only a movement of the foot, while a prick may cause a movement of the whole limb. At the same time there is much more than degree involved in the difference between these two stimuli. The impression that is felt as pain causes a quick simple movement of the part, in which chiefly the flexors are involved. It is a movement obviously to withdraw from harm, and it is proportioned, in energy and extent, to the intensity of the stimulation. To this, however, there is one limitation:—a strong impression of pain may prevent all reflex movement by exerting an inhibitory influence on the centres. On the other hand, a tactile impression causes a much more complex movement, which does not bear a simple relation, in extent and energy, to the degree of the sensory impulse, but is also influenced by the quality of the impression at the place at which it is produced. Thus the reflex movement produced from the palm or sole is especially elaborate; it may present an indication of the central relations by which the purposed use of the extremities is facilitated. The effect of all painful impressions is similar, whether these are produced through the nerves of the skin or those of deeper structures. The reflex action caused by pinching

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Although no other part of the *alimentary canal* is under voluntary influence, all parts are probably related to the spinal cord, by means of the connection between the sympathetic system and the nerve-roots. Constipation is extremely common in disease of the spinal cord, and is often greater than can be accounted for by the mere loss of power of the abdominal muscles. The pain in the back that is so common in disease of the stomach has been regarded as a reflected pain, due to this connection.

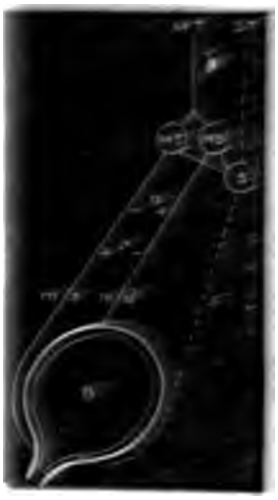

FIG. 84.—DIAGRAM SHOWING PROBABLE PLAN OF THE CENTRE FOR MICTURITION.
MT, motor tract; ST, sensory tract in the spinal cord; MS, centre, and *ms*, motor nerve for sphincter; MD, centre, and *md*, motor nerve for detrusor; *s*, afferent nerve from mucous membrane to S, sensory portion of centre; B, bladder. At *r* the condition during rest is indicated, the sphincter centre in action, the detrusor centre not acting. At *a* the condition during action is indicated, the sphincter centre inhibited, the detrusor centre acting.

The *uterine* functions are probably independent of the cord in greater degree than those of any other organs under the influence of the sympathetic. The function of menstruation goes on independently of cord disease, and may be regular although there is absolute arrest of all conduction, sensory and motor. Pregnancy and labour may proceed in normal course, in spite of such disease of the cord as causes complete motor paraplegia.

Sexual Functions.—The activity of the sexual organs depends on the integrity of the reflex loop to and from a special centre, also situated in the lumbar enlargement, but the due action of this centre depends on cerebral (psychical) as well as on reflex influences. Disease of the centre, or of the nerves leading to or from it, abolishes sexual action. The sexual reflex is, however, one of the cutaneous reflexes, and it shares the condition of these rather than of the muscle-reflex processes.

The centre is probably double, and its action is impaired by interference with either half. When, by disease higher up, the connection with the psychical centres is interrupted, the sexual act cannot be perfectly performed. If the path from the controlling centre (p. 20) shortened adapt themselves to the change as it proceeds. Apparently the increased tension on the former makes them yield to the movement, and the lessened tension on the latter makes them contract, each set thus preserving its proper tone. The tone of a muscle seems to be produced by a reflex process, due to a certain state of the motor cells regulated by the impressions from the afferent muscle-nerves. These, as we have seen, are produced by two mechanical influences, the lateral pressure of the fibres (as in the pain of cramp) and the tension on the muscle. An increase of the latter, in passive movement, seems to lessen the activity of the cells and permit the fibres to elongate; a diminution increases the activity of the cells and causes the fibres to shorten. Whether any other afferent impressions influence the process we do not know.

Co-ordination of Movement.—The co-ordination of muscular contractions, which results in the ordination or ordering or arrangement of movement, has been already described (p. 7), so far as its general features are concerned. We cannot ascribe to the spinal cord any process that can be designated the arrangement of movement; since the muscular contractions correspond to the activity of the motor cells of the cerebral cortex and to the corresponding impulses along the pyramidal fibres. These determine the initial activity of the motor cells of the cord both for the muscles that move a part, and for those that oppose the action of the others and give steadiness and uniformity to the movement. But these impulses from the brain descend on spinal cells that are not in a passive, inactive, functionless state. Such a state probably never exists, for even when there is no voluntary activity, there must be the activity on which the tone depends, that, as we have just seen, must exist in all postures of the limbs. This state of the cells preceding voluntary activity is determined by the afferent impulses, chiefly by those from the muscles. Through these the muscles are adapted to posture, and in the mechanism for adaptation to posture we may trace also a mechanism for part at least of the co-ordination effected in the spinal cord. This seems to consist of two parts. (1) An automatic variation in the state of muscles, permitting the ordered performance of intended movements. The state of the afferent impulses acting from the muscle on the motor cells must be at once altered by the effect of the voluntary activity of the muscles, since this will alter the impulses in the muscle-nerves by the changed and changing pressure and tension on the tissues in which these nerves end—the pressure of the contracting and widening fibres and the tension in the antagonists that oppose the motion, but do not prevent it, and are made more tense by the muscles they are opposing. These impulses pass to the cord, and there act on the motor cells, and influence their state in a way that we can at present only guess at, but which must be of the utmost importance. There must of necessity be a definite relation between certain states of the muscle and the

Table showing the approximate relation to the spinal nerves of the various motor, sensory, and reflex functions of the spinal cord.

MOTOR.	(NERVES.)	MOTOR.		SENSORY.		REFLEX.
	C 1	Small rotators of head Depressors of hyoid	1	Scalp.	1	
St.-mastoid, Upper neck muscles, Upper part of Trapezius	2		2	Neck and upper part of chest	2	
	3	Lev. ang. scapulæ	3		3	
	4	Diaphragm Shoulder muscles	4	Shoulder	4	
	5	Serratus Flex. of elbow Supinators	5	Arm, outer side	5	Scapular
Lower neck muscles, Middle part of Trapezius	6	Ext. wrist & fingers	6	Radial side, forearm and hand; thumb	6	
	7	Ext. elbow Flex. wrist & fingers Pronators	7		7	
	8	Muscles of hand	8	Arm, inner side; ulnar side of forearm and hand; tips of fingers	8	
	D 1	Intercostals	1		1	
Lower part of Trapezius and Dorsal muscles	2		2	Front of thorax	2	
	3		3		3	
	4		4		4	Epigastric
	5		5		5	
	6		6	Ensiform area	6	
	7	Abdominal muscles	7		7	
	8		8	Abdomen (Umbilicus 10th)	8	Abdominal
	9		9		9	
	10		10		10	
	11		11		11	
	12		12	Buttock, upper part	12	
	L 1		1	Groin and scrotum (front)	1	Cremasteric
Lumbar muscles	2	Cremaster Flexors of hip	2	Thigh: outer side front inner side	2	*Knee-jerk*
	3	Extensors of knee Adductors of hip	3		3	
Peroneus, L. Flex. of ankle, Ext. of ankle	4	Ext. & abduct. of hip	4		4	Gluteal
	5	Flexors of knee	5	Leg, inner side	5	*Foot-clonus*
	S 1	Intrinsic muscles of foot	1	Buttock, lower part Back of thigh Leg & foot except inner part	1	Plantar
	2		2		2	
	3	Perineal and anal muscles	3		3	
	4		4	Perinæum and anus	4	
	5		5	Skin from coccyx to anus	5	
	Co.		Co.		Co.	

resulting movement.* The impulses may reach the cerebellum, as we have seen, from the grey matter of the post-pyramidal nucleus in which these fibres end.† But this primary co-ordination is not a function of the cord, although it may be lost in consequence of disease of the cord. The direct cerebellar tract may constitute a similar path, but of this we have only the evidence of destination and analogy.

It is probable that the mechanisms just alluded to, as constituted by the functional relations between the motor and sensory structures in the cord, very much facilitate the execution of many movements, and especially of those that are the more simple in their character. The less variation a movement is capable of, or needs, the more largely may the spinal mechanism be concerned in its arrangement; and hence such actions as standing and walking may be effected through a comparatively small number of the pyramidal fibres, and, as we shall see, they may still be performed when there is disease that would prevent a more elaborate action. It is also probable that in animals such spinal mechanisms are developed to a greater extent than in man, and care must be taken in drawing inferences from one to the other.

The impulses from the muscles that determine the muscle-reflex action and the spinal co-ordination, must pass by nerves that end in the grey matter and are connected with nerve-cells, at or near the level at which they enter the cord. We do not know whether it is through these fibres, or through those that pass upwards without interruption, that the impulses are conveyed which, when excessive in degree, give rise to muscular sensations. But attention may again be called to the important evidence, afforded by such sensations, of the abundance of upward impulses of which we are normally unconscious. These nerves, it will be remembered, begin in the connective tissue of the muscles, and the acute pain of cramp, and the muscular tenderness in such affections as multiple neuritis, afford indirect evidence of impulses which we do not usually observe, and might almost doubt; while the manner in which such pain is excited is very clear evidence of the sensitiveness of the nerves to the modes of stimulation that have been assumed as effective. We have already seen (p. 9) that it is through such unfelt impulses that we probably derive the chief part of our conceptions of posture, a subject that must again be considered in connection with the brain.

INFLUENCE ON NUTRITION.—The nutrition of the *muscles* is under the control of the anterior grey matter, and probably of the motor nerve-cells. The influence is exerted through the motor nerves, and indeed consists in the normal integrity of the fibres (see p. 24).

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* This has been experimentally confirmed by Bechterew, who finds that section of the posterior columns, in various animals, above the lumbar enlargement causes a defect of co-ordination similar to that produced by loss of the cerebellum (Bechterew, 'Wjestnik psich. i. nevropat.,' 1889, vii, 1; and 'Neur. Centralbl.,' 1890, p. 82).

† A connection recently confirmed by Brosset, 'Des Connexions du Cervelet,' Paris, 1891.

The nutrition of the *bones* and *joints* is also under the control of the cord, and probably is exerted through the posterior roots. It is true, the growth of the bones is hindered when the anterior cornua are diseased, but no atrophic change seems then to occur in the osseous structure, and the influence of the paralysis may be indirect. A true trophic change, and an alteration in the joints, may occur in locomotor ataxy, in which the disease is of the posterior nerve-fibres.

The nutrition of the *skin* seems also to depend on nerves that have their course in the posterior roots. The clearest fact is that irritation of the nerve-structures has far more effect than simple loss of function. It is when the cord or nerve-roots are the seat of irritative inflammation that the most acute trophic changes occur, while mere degeneration of these nerves induces slower and slighter changes (see p. 22).

VISCERAL CONTROL.—Although the viscera are under the immediate control of the sympathetic system of nerves, they are related to centres in the spinal cord, and it is from these centres that the controlling influence is really derived, probably by means of the finer fibres of the anterior roots. The relation is the most direct and important, so far as concerns the diseases of the spinal cord, in the case of the organs over which the will has an influence, the *rectum* and the *bladder*. The centres for these are in the lumbar enlargement, but we do not know their exact position. They are probably complex reflex centres. We can best understand their action by studying them when voluntary influence is lost.

The centre for the sphincter ani is the more simple, but the system of action of each is probably similar. In the wall of each viscus we have muscular fibres to expel the contents, and at the mouth a sphincter arrangement to prevent continuous evacuation. Fæces or air in the rectum, and urine in the bladder, may excite the lumbar centre, and cause two effects—contraction in the wall and relaxation of the sphincter. This process can be controlled by the will to a considerable extent, although we are still ignorant of the precise mode in which the voluntary influence is exerted. But if the volitional path in the cord is interrupted above the lumbar centres, the will can no longer control the reflex processes; as soon as fæces irritate the rectum, they are expelled by the reflex mechanism; as soon as a sufficient quantity of urine accumulates in the bladder, a reflex contraction of the detrusor and relaxation of the sphincter cause its escape. The affection of the voluntary path for the sphincter is not always proportioned to that for the legs. If the damage to the cord involves also the sensory tract, the patient is unconscious of the action of the bladder or bowel. If the sensory tract is unaffected, the patient is aware of the process, but cannot control it. It is often said that there is permanent relaxation of the sphincters, but this is true only when the lumbar centres are inactive or destroyed. In this condition evacuation occurs as soon as fæces or urine enter the bowel or ~~rectum~~ bladder. The urine escapes continuously, instead of being expelled at intervals. The condition is less obvious in the case of the rectum, because there is no such continuous passage of fæces into the rectum as there is of urine into the bladder. We may, however, distinguish between the two states of the rectum by the introduction of the finger. If the lumbar centre is inactive, there is a momentary contraction, due to local stimulation of the sphincter, and then permanent relaxation. If, however, the reflex centre and motor nerves from it are intact, the introduction of the finger is followed, first by relaxation, and then by gentle, firm, tonic contraction. I have verified this by introducing an india-rubber cylinder instead of the finger, and registering the pressure on the cylinder by connecting it with a recording apparatus. The relaxation is then found to be preceded by a very slight brief contraction, and to be followed by unbroken tonic contraction. The relaxation may also be readily produced by any impression on the mucous membrane above the sphincter. Fig. 83 shows some of the tracings obtained.

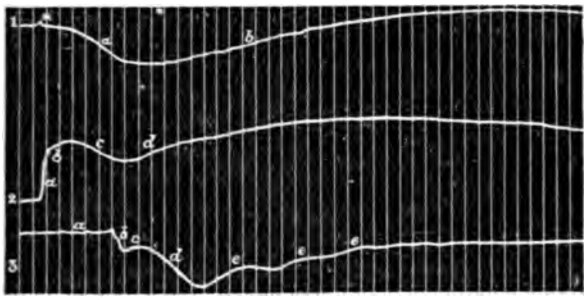

Fig. 83.—Tracings of the Action of the Sphincter Ani.
The vertical lines represent seconds of time.

1. Effect on contraction of sphincter of the injection into the rectum of a small quantity of air at *. *a*, fall in pressure due to the inhibition of the contraction; *b*, rise due to the slowly returning contraction. 2. Effect of the introduction of the instrument. *a*, sudden rise of lever at moment of introduction, due to the exposure of the instrument to the pressure of the sphincter (the top of this line represents the degree of previous contraction); *b*, initial rise due to increased contraction; *c*, fall from partial inhibition; *d*, subsequent contraction, rising to a greater degree than the initial contraction, and subsequently falling slightly. 3. Effect of cough. *a*, pressure of tonic contraction of sphincter (the slight irregularities are due to pulse-waves); *b*, fall in pressure, due to the movement of the instrument by the cough; *c*, initial contraction; *d*, relaxation of inhibited sphincter; *e*, *e*, *e*, rhythmical variations in subsequent rise. (From the 'Proc. Royal Society,' 1877.)

The action of the bladder mechanism can be best understood by assuming that the motor centre really consists of two parts, one (MS, Fig. 84) maintaining the contraction of the sphincter, the other (MD) exciting the contraction of the detrusor fibres, and that these two parts are antagonistic; when one acts the other is inhibited. Thus,

nerve-cells. The influence is exerted [illegible] the detrusor at rest.
indeed consists in the normal integrity of [illegible] from the bladder and a

The nutrition of the *bones* and *joints* is [illegible] centre acts, and
cord, and probably is exerted through th[illegible] power is impaired,
the growth of the bones is hindered w[illegible] insufficient, and then
diseased, but no atrophic change seems [illegible] centre may yield too
structure, and the influence of the paraly[illegible] incontinence.
trophic change, and an alteration in the [illegible] is under voluntary
ataxy, in which the disease is of the pos[illegible] spinal cord, by means

The nutrition of the *skin* seems also to [illegible] between the sympa-
their course in the posterior roots. The [illegible] the nerve-roots. Con-
of the nerve-structures has far more effe[illegible] common in disease
It is when the cord or nerve-roots are [illegible], and is often greater
mation that the most acute trophic cha[illegible] for by the mere
ration of these nerves induces slower an[illegible] the abdominal muscles.

Visceral Control.—Although the [illegible] back that is so common
control of the sympathetic system of ne[illegible] the stomach has been re-
in the spinal cord, and it is from thes[illegible] pain, due to this
influence is really derived, probably [illegible]
the anterior roots. The relation is th[illegible] functions are probably in-
so far as concerns the diseases of [illegible] the cord in greater degree
the organs over which the will has a[illegible] any other organs under
bladder. The centres for these are in [illegible] the sympathetic. The
do not know their exact position. Th[illegible] menstruation goes on inde-
centres. We can best understand the[illegible] cord disease, and may be
voluntary influence is lost. [illegible] there is absolute arrest

The centre for the sphincter ani is [illegible], sensory and motor.
of action of each is probably similar. [illegible] labour may proceed in
muscular fibres to expel the content[illegible], in spite of such disease
arrangement to prevent continuous [illegible] causes complete motor
rectum, and urine in the bladder, [illegible]
and cause two effects—contraction in [illegible] *Functions.*—The activity of
sphincter. This process can be contr[illegible] organs depends on the in-
able extent, although we are still igno[illegible] the reflex loop to and from
the voluntary influence is exerted. [illegible] centre, also situated in the
cord is interrupted above the lumba[illegible] enlargement, but the due action
control the reflex processes; as soon [illegible] centre depends on cerebral
are expelled by the reflex mechanism [illegible] as well as on reflex influences.
of urine accumulates in the bladder, [illegible] the centre, or of the nerves
and relaxation of the sphincter caus[illegible] or from it, abolishes sexual
voluntary path for the sphincter is [illegible] The sexual reflex is, however,
for the legs. If the damage to the [illegible] shares the condition of these
the patient is unconscious of the [illegible].
the sensory tract is unaffected, the [illegible] its action is impaired by inter-
cannot control it. It is often said [illegible] disease higher up, the connection
of the sphincters, but this is true o[illegible], the sexual act cannot be
inactive or destroyed. In this c[illegible] the controlling centre (p. 20)

the muscular action is more or less paroxysmal and varying, and the muscles are at times extended by their opponents. Thus in paroxysmal extensor spasm in the legs, no shortening occurs in the muscles moving the knee, because they are equally balanced, but the calf muscles, being stronger than the flexors of the ankle, contract the more, and extend this joint, whenever a paroxysm of spasm comes on, and, after a time, may undergo slight structural shortening, so that the foot cannot be flexed on the leg beyond a right angle.

It is of great importance to distinguish the shortening of muscles due to tissue changes in them, from the active contracture that may closely simulate it. In the latter, gentle extension, kept up for a few minutes, restores the muscle to its normal length. The importance of the distinction is due to the fact that the structural shortening can be removed by tenotomy or forced extension, while these measures are useless in the case of active contracture, which would subsequently reassert itself.

Inco-ordination of movement, although motor, depends usually on disease of afferent fibres. Its characters have been already described in outline (p. 7).

It occurs in three forms, of which the third differs entirely from the others in aspect and nature, and alone is a true motor symptom. The two chief forms are the following :—(1) Simple disorder of voluntary muscular contractions, which, when considerable, amounts to a wild irregularity of movement, and when slight is only manifested by slight irregularity when the guidance of vision is withdrawn—as in the inability to maintain the muscular contractions in the perfect uniformity needed for equilibrium when the base of support is rendered small by the juxtaposition of the feet. The delicacy of the test is increased by the removal of the firm base afforded by the boot, and by closure of the eyes. The increase in unsteadiness when the eyes are closed is sometimes termed "Romberg's symptom." This condition results from interruption of the afferent path from the muscles to the spinal cord, including the fibres that ascend the cord and those that subserve the muscle-reflex process at the level of entrance. The effect of the loss of the latter is a cessation of the chief part of spinal co-ordination, which, as we have seen, depends on those processes, co-operating with the voluntary impulse. Hence it is associated with loss of the knee-jerk. The loss of other forms of reflex action does not seem capable of causing inco-ordination. In complete interruption of the root-fibres from the muscles the mechanism of the next form is necessarily conjoined. (2) Inco-ordination resembling the slighter degrees of that just described, and consisting especially in such a defect of equilibrium as results from disease of the middle lobe of the cerebellum, is produced by disease of the posterior median columns above the lumbar enlargement, or, if it extends into the latter, leaving the root-zones free so as not to impair muscle-reflex action. This is apparently produced by interruption of the path by which impressions from the muscles are conveyed to the posterior nuclei of the medulla and probably thence to the cerebellum (p. 205). The effect is to deprive the motor cortex of the guidance afforded by these impulses. The difference between this form and the slighter degrees of the first is that the muscle-reflex action is not lost, the knee-jerk being preserved. (3) Irregular compression of the motor fibres in the cord apparently causes the peculiar jerky inco-ordination seen in disseminated sclerosis, which is supposed to depend on unequal and irregular conduction along the fibres, due to their compression. This form, however, needs further study.

SENSORY SYMPTOMS.—Loss of sensation is a very common effect of disease of the spinal cord. It may be total, and involve all forms of sensibility, or partial, and affect only some forms. The statements made on pp. 8—10 regarding impairment of sensation generally, and the method of ascertaining it, apply to that which results from cord disease, and need not be here repeated. It has been also pointed out (p. 60) that loss of sensation occurs less readily than loss of motor power. In cord disease it is common for muscular paralysis to exist with intact sensibility. This may partly depend on the fact, ascertained by experiments on the effect of local anæmia of the cord, that the sensory fibres, of the nerve-roots at least, seem to have more resisting power than the motor fibres. Sensation may be impaired by disease of any part of the sensory path,—posterior roots, probably also the posterior cornua and commissure, or the conducting tracts up the cord. A division of the sensory path into upper and lower segments is conceivable, analogous to that of the motor path, although we have not the same clear ground for the distinction into simple segments of similar composition.

Disease of the posterior nerve-roots causes loss of reflex action as well as impairment of sensation, just as disease of the anterior roots interferes with reflex action as well as with motor power. Interruption of the sensory conducting tracts higher up leaves reflex action unaffected. But a focal lesion, such as transverse myelitis, may damage both the conducting tracts and the nerve-roots at the level of the lesion, or may affect only one of these. Disease outside the cord, compressing it, may have the same double effect. Hence it is important to test sensation at the level of the lesion, as well as in the parts below, and to remember that the "level of the lesion" may involve the limbs or the trunk. Areas of anæsthesia may thus be found on the trunk when there is none on the legs, and may be of considerable diagnostic importance, *e. g.* in spinal caries.

Random disease of the nerve-roots outside the cord usually impairs all forms of sensibility, although slight damage may arrest the conduction of tactile impressions, and not those of pain, which are probably more energetic. Disease of the roots within the cord often causes only partial loss, because the fibres which conduct different impressions have a different course. But loss of one form of sensibility from disease of the root-fibres or nerve-fibres usually depends on the kind of disease, and is especially due to degenerative changes, the result of some present or past local influence. Disease higher up the cord still more frequently causes partial loss; either sensibility to pain or to touch may be impaired. That to temperature is rarely affected without that to pain. We do not yet know precisely the significance of this special form of loss, because, as we have seen, we are still uncertain as to the path for each form of sensibility. It is highly probable, however, that loss of sensibility to pain is produced by disease of the antero-lateral ascending tract.

Disease of the posterior median column, and possibly that of the cerebellar tract, certainly impairs the conduction of impressions from the muscles. It probably abolishes the "muscular sense" of posture and movement, although this does not entail a distinct sensory loss. But there may be also a greater loss of muscular sensibility, so that the normal sensitiveness to pressure and passive extension (and even at last to electrical stimulation) may be impaired or lost. The "common sensibility" of muscles is probably lost only when the disease of the nerves is greater than suffices to abolish what is termed the "muscular sense." Interference with this path in the nerves or roots outside the cord abolishes muscle-reflex action, and also causes more pronounced symptoms (ataxy, &c.) than disease higher up the path within the cord.

Increased sensitiveness, hyperæsthesia and hyperalgesia, are also common in disease of the spinal cord, and usually depend on irritation of the conducting fibres in some part of their course. Probably the irritation produces the effect by intensifying the impulse as it passes, since the phenomena of stimulation of nerves show that their axis-cylinders have some power of transforming other forms of energy into nerve-force, *i. e.* of evolving nerve-force, and therefore of increasing the strength of that which passes along them.

The term "hyperæsthesia" is commonly used in the sense of "hyperalgesia." In the strict sense of the word, hyperæsthesia is seldom observed or even searched for.

When the sensibility to touch is "increased" there is some perversion of the sensation, not a simple increase. The sensation may be felt as "thrilling" or "shock-like sensations." When there is an extreme increase, pain may be produced by a touch, but it is more probable that the touch stimulates the over-sensitive nerves of common sensibility, than that actual pain is produced through the tactile nerves. A touch may cause pain when it is not felt as a touch, as in the condition termed "anæsthesia dolorosa." Other forms of altered sensibility have been already described.

Pain, referred to the spine, occasionally present in organic disease of the cord, is more frequent in disease originating in the meninges or bones. But the frequency with which spinal pain is present in abdominal, especially gastric, disease, and in neuralgic affections, depend on damage to the central structures in the cord and nerve-roots, which are present only at the level of that damage, *i. e.* in the parts functionally related to that part of the cord. The two sets of symptoms may be distinguished as "conducting" and "central;" but in many diseases those at the level of the lesion are chiefly due to the irritation of the nerve-roots, and these are most usefully designated "root symptoms."

Disease of the cord does not always cause bilateral symptoms. A little consideration will show that this must be so. A random process, of small extent, may damage one side only. It may affect one or several structures on one side or all of them. Nevertheless it is very rare for such a process to spare the other side altogether, and practically no lesion affects all parts of one side without damaging, in some degree, the other side, at any rate for a time. In very rare forms of traumatic lesion, however, the damage to the other side is almost imperceptible. Again, "system diseases," in quite exceptional cases and for unknown reasons, are limited to one side; much more frequently they may affect one side earlier and more than the other. Such unilateral symptoms have been awkwardly termed "hemi-paraplegia."

Motor Symptoms.—*Loss of motor power* results from interference with the motor path in any part of its intra-spinal course,—pyramidal tracts, anterior grey matter, anterior nerve-roots. We have seen that the path may be divided into two segments, upper and lower (see p. 176). The spinal cord contains a considerable portion of the nerve-fibres of the upper segment, and their lower termination in the grey matter, but only the commencement of the lower segment, the motor ganglion-cells, and root-fibres proceeding from them. The chief part of the lower segment is outside the spine, in the nerve-trunks. So far as the loss of power is concerned the effect is the same, in whatever part of the motor path the interruption is situated; but the other symptoms that accompany the loss of power differ very much according as the interruption is in the upper or lower segment. The lower segment influences muscular nutrition, and forms part of the path of reflex action. Hence, as we shall presently see, the muscles waste, and reflex action is lost, when this is diseased; but when the upper segment is damaged there is no muscular wasting, and reflex action, instead of being lost, is commonly increased. *The symptoms produced by disease of either segment are essentially the same, whatever part of the segment is diseased, whether the cell at the upper extremity, the fibre, or the ramification at the lower extremity of the fibre.* If, for instance, the intra-muscular nerve-endings are paralysed by curara the symptoms produced are the same as if the fibres in the nerve-trunks are divided or these cells in the cord are destroyed. Disease of the termination of the upper segment in the grey matter of the cord must, of necessity, produce the same effect as disease of the pyramidal fibre itself, or the cell in the cerebral cortex from which the fibre springs.

We shall afterwards see that this consideration has very important applications.

If a fibre of either segment is seriously damaged, it degenerates below the division, since its nutrition depends on the influence of the cell from which it has sprung. If damaged only by pressure, however complete may be the evidence of degeneration, regeneration is possible, even after one or two years. It is possible that such regeneration also occurs when the damage is by inflammation. The nutritional stability of the fibre, or rather of its essential element, the axis-cylinder, becomes less, and more easily deranged, the greater the distance from the parent cell, and it is least in the terminal ramification of each segment. This is probably the reason why curara acts chiefly on the intra-muscular nerves, and it explains the influence of many poisons on the peripheral nerve-endings, and the facts of nerve-degeneration in tabes and "multiple neuritis," &c. The law is probably also true of the upper segment; and if so, many facts of pathology become clearer to us, as will be seen in the chapter on "spastic paraplegia." It may thus be taken as a general law of the utmost importance that *the vitality of a nerve-fibre and its power of resisting morbid influences are less the greater the distance from the cell from which it springs and of which it is really part.*

A lesion of the pyramidal tract causes loss of power in all parts below the level of the disease—that is to say, of all parts the fibres for which are interrupted. A lesion of the grey matter, or of the anterior roots, causes paralysis only of those parts which are functionally on the level of the lesion. The two mechanisms often coincide. A transverse lesion in the cervical enlargement, for instance, may cause paralysis of the arms from the damage to the grey matter and nerve-roots, and paralysis of the legs from damage to the pyramidal fibres. The disease involves, primarily in the former case, the commencement of the second segment of the motor path for the arms; in the latter, the middle of the upper segment. The associations of the paralysis differ accordingly. The parts affected by a lesion at any level will be readily ascertained by an examination of the table of functions on p. 210.

It is important to note that the affection of motor power is often incomplete. It may then involve one set of muscles more than another. The flexors or the extensors may be chiefly paralysed. In disease of the dorsal cord, it is very common for the flexor muscles of the hip and knee to suffer more than the extensors. Why this should be we do not know, but the fact is important, because considerable weakness of the flexors of the knee is readily overlooked. In examining motor power, each set of muscles should be separately tested.

Motor over-action; Spasm is frequent in disease of the spinal cord and its membranes. It may present the form of *tonic* spasm, which when persistent is termed *rigidity*, or of *clonic* spasm, which is usually transient and paroxysmal. Tonic spasm, persistent, and involving only a certain group of muscles, causes distortion of the parts to which they are attached, and is often termed *contracture.* Tonic spasm, paroxysmal or persistent, is far more frequent than clonic spasm, which occurs chiefly in association with tonic spasm as the muscular clonus described at p. 222, or as a consequence of certain functional derangements. Tonic spasm may be the result of direct irritation of the motor structures, or may depend on over-action of the reflex centres, due, not to irritation, but to deficient control. Either form may be acute or chronic. The acute irritation which causes spasm is generally inflammation of the membranes, rarely an acute lesion of the substance of the cord. The chronic irritation is chiefly due to compression of the motor fibres—either of the pyramidal tracts, when there may be persistent unvarying spasm in the parts below the disease, related to the fibres irritated,—or of the nerve-roots, when similar spasm is produced in the parts at the level of the lesion. The spasm that depends on reflex action is seen chiefly in the legs and trunk, but especially in the former. It may preponderate in either the flexors or extensors, so that the legs, when rigid, may be drawn up or straight out. The flexor spasm seems to be due to an over-action of the centres for cutaneous reflex action, the extensor spasm chiefly to that of the centres for muscle-reflex action, although it may also be excited indirectly by a cutaneous impression. This form will be considered in connection with the reflex over-action.

Muscular Contraction.—The actual shortening of muscles, by which they cannot be passively extended to their normal length, is due to tissue changes fixing the active persistent tonic spasm or contracture just mentioned. The rapidity with which it occurs depends on the uniformity of the spasm, and we may distinguish three modes in which it arises, which differ in the time required and degree attained. (1) When one set of muscles is paralysed, their opponents, never being extended, quickly become fixed in the contracted condition which they assume in virtue of their power of adaptation to posture. Thus in palsy of the flexors of the ankle, the calf muscles quickly become contracted, so that the foot cannot be flexed on the leg even up to a right angle. The same thing happens by a similar mechanism, when one posture is unceasingly maintained and one set of muscles are never extended, *e. g.* persistent flexion of the knee leads quickly to structural shortening of the hamstring muscles. (2) Where there is unchanging active contracture from chronic irritation of the motor fibres by a growth, or permanent reflex contracture, as when reflex flexor spasm keeps the hip and knee permanently flexed, similar structural changes occur, so that passive elongation soon becomes impossible. (3) In paroxysmal tonic spasm of intense degree, if one set of muscles is stronger than their opponents, their action may so preponderate as to lead to a greater active contracture than their opponents present, and permanent shortening may occur; although it does so slowly and to a slighter degree than in the other forms, because the muscular action is more or less paroxysmal and varying, and the muscles are at times extended by their opponents. Thus in paroxysmal extensor spasm in the legs, no shortening occurs in the muscles moving the knee, because they are equally balanced, but the calf muscles, being stronger than the flexors of the ankle, contract the more, and extend this joint, whenever a paroxysm of spasm comes on, and, after a time, may undergo slight structural shortening, so that the foot cannot be flexed on the leg beyond a right angle.

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Sensory Symptoms.—Loss of sensation is a very common effect of disease of the spinal cord. It may be total, and involve all forms of sensibility, or partial, and affect only some forms. The statements made on pp. 8—10 regarding impairment of sensation generally, and the method of ascertaining it, apply to that which results from cord disease, and need not be here repeated. It has been also pointed out (p. 60) that loss of sensation occurs less readily than loss of motor power. In cord disease it is common for muscular paralysis to exist with intact sensibility. This may partly depend on the fact, ascertained by experiments on the effect of local anæmia of the cord, that the sensory fibres, of the nerve-roots at least, seem to have more resisting power than the motor fibres. Sensation may be impaired by disease of any part of the sensory path,—posterior roots, probably also the posterior cornua and commissure, or the conducting tracts up the cord. A division of the sensory path into upper and lower segments is conceivable, analogous to that of the motor path, although we have not the same clear ground for the distinction into simple segments of similar composition.

Disease of the posterior nerve-roots causes loss of reflex action as well as impairment of sensation, just as disease of the anterior roots interferes with reflex action as well as with motor power. Interruption of the sensory conducting tracts higher up leaves reflex action unaffected. But a focal lesion, such as transverse myelitis, may damage both the conducting tracts and the nerve-roots at the level of the lesion, or may affect only one of these. Disease outside the cord, compressing it, may have the same double effect. Hence it is important to test sensation at the level of the lesion, as well as in the parts below, and to remember that the "level of the lesion" may involve the limbs or the trunk. Areas of anæsthesia may thus be found on the trunk when there is none on the legs, and may be of considerable diagnostic importance, *e. g.* in spinal caries.

Random disease of the nerve-roots outside the cord usually impairs all forms of sensibility, although slight damage may arrest the conduction of tactile impressions, and not those of pain, which are probably more energetic. Disease of the roots within the cord often causes only partial loss, because the fibres which conduct different impressions have a different course. But loss of one form of sensibility from disease of the root-fibres or nerve-fibres usually depends on the kind of disease, and is especially due to degenerative changes, the result of some present or past local influence. Disease higher up the cord still more frequently causes partial loss; either sensibility to pain or to touch may be impaired. That to temperature is rarely affected without that to pain. We do not yet know precisely the significance of this special form of loss, because, as we have seen, we are still uncertain as to the path for each form of sensibility. It is highly probable, however, that loss of sensibility to pain is produced by disease of the antero-lateral ascending tract.

Disease of the posterior median column, and possibly that of the cerebellar tract, certainly impairs the conduction of impressions from the muscles. It probably abolishes the "muscular sense" of posture and movement, although this does not entail a distinct sensory loss. But there may be also a greater loss of muscular sensibility, so that the normal sensitiveness to pressure and passive extension (and even at last to electrical stimulation) may be impaired or lost. The "common sensibility" of muscles is probably lost only when the disease of the nerves is greater than suffices to abolish what is termed the "muscular sense." Interference with this path in the nerves or roots outside the cord abolishes muscle-reflex action, and also causes more pronounced symptoms (ataxy, &c.) than disease higher up the path within the cord.

Increased sensitiveness, hyperæsthesia and hyperalgesia, are also common in disease of the spinal cord, and usually depend on irritation of the conducting fibres in some part of their course. Probably the irritation produces the effect by intensifying the impulse as it passes, since the phenomena of stimulation of nerves show that their axis-cylinders have some power of transforming other forms of energy into nerve-force, *i. e.* of evolving nerve-force, and therefore of increasing the strength of that which passes along them.

The term "hyperæsthesia" is commonly used in the sense of "hyperalgesia." In the strict sense of the word, hyperæsthesia is seldom observed or even searched for.

When the sensibility to touch is "increased" there is some perversion of the sensation, not a simple increase. The sensation may be felt as "thrilling" or "shock-like sensations." When there is an extreme increase, pain may be produced by a touch, but it is more probable that the touch stimulates the over-sensitive nerves of common sensibility, than that actual pain is produced through the tactile nerves. A touch may cause pain when it is not felt as a touch, as in the condition termed "anæsthesia dolorosa." Other forms of altered sensibility have been already described.

Pain, referred to the spine, occasionally present in organic disease of the cord, is more frequent in disease originating in the meninges or bones. But the frequency with which spinal pain is present in abdominal, especially gastric, disease, and in neuralgic affections, lessens its significance when it exists alone. In meningitis, acute or chronic, and in meningeal growths, spinal pain is frequent, and in organic disease of the bones of the vertebral column it is an almost constant symptom, and is combined with local tenderness. The same combination of local pain and tenderness frequently occurs in cases of neuralgic pain, "rachialgia,"—a condition that is often loosely termed "spinal irritation," especially when it succeeds, as it often does, concussion of the spine. Such pain is usually felt through a considerable extent of the vertebral column, or has more than one place of chief intensity and tenderness. That which is due to organic disease is usually fixed and unchanging. In organic disease of the cord itself, pain is more often referred to the neighbourhood of the spine, to the loins or the sacrum, than to the spinal column itself.

Still more important are the pains that are referred to the parts to which the sensory nerves are distributed, and have hence been termed "excentric" or "radiating" pains. They are of two kinds: (1) those due to the irritation of the posterior nerve-roots in their passage through the intervertebral foramina, through the membranes, or through the posterior columns of the cord; (2) those produced by irritation of the sensory conducting tracts. The pains of the first class are called "root-pains," and are often intense. They correspond in level to the disease. Those of the second class are sometimes acute, especially in lesions that irritate the conducting tracts by pressure, *e.g.* growths. More often they are dull pains, closely resembling rheumatism, and frequently mistaken for rheumatism by the patients themselves and their medical attendants. The mistake is the more easily made, because other symptoms suggestive of spinal disease may be inconspicuous, and the rheumatoid pains in chronic cases may be influenced by weather, being much more troublesome in damp and cold than in fine and dry weather. In all cases, persistent rheumatic pains in the limbs should suggest the possibility of spinal disease, and watch should be kept for such symptoms as local loss of power, or alterations in reflex action. There is a third class of pains—resembling root-pains, which depend on degenerative changes in the nerve-fibres; the molecular alterations that result give rise to upward impulses of considerable intensity. They are met with in locomotor ataxy and in multiple neuritis, and may be dull as well as acute. The position in which these various radiating pains are felt—legs, trunk, or arms—depends upon the seat of the disease. Although often produced in the root-fibres, these pains may also be due to the peripheral nerves.

It is especially important to note that the root-pains are frequently felt as a sense of constriction, a painful sense of tightness, as if a band were tied tightly around the part—the "girdle-pain," as it is called. When there is transverse damage to the cord, at the lowest part of the healthy region there is a state of irritation of the sensory nerves, and this irritation (referred to the nerve-endings) causes the girdle-pain. It is named from its frequency at the level at which a girdle is worn, the middle of the trunk, which depends on the dorsal region being the most common seat of disease; but it may be felt lower down, about the groins or even the legs, or higher up, in the arms. In the limbs it is a mere sense of tightness.

The girdle-pain is a symptom chiefly of inflammatory and degenerative processes within the cord. Pressure on the nerve-roots usually causes acute pain, but the most severe root-pains are those met with in disease of the bones of the spine. These have also, more than any other kind, the characteristic that they are increased by movement, and in growths commencing in the bones (which are usually malignant) this feature is of considerable diagnostic importance. The suffering in such cases is so great as to have gained for the resulting symptoms the ominous designation of paraplegia dolorosa.

Spontaneous sensations, other than pain, are very common in disease of the spinal cord, and are often of considerable diagnostic importance, but suggestive rather of the presence of a morbid state than of its nature. They may present many varieties of character, but the familiar sensation produced by pressure on a nerve-trunk, when the part is said to be "asleep," or to have "pins and needles," is the most common and the most significant. It may occur in functional disturbance of the cord as well as in organic disease, and also is very common in peripheral neuritis. It depends on over-action of the sensory structures, but it has at present no special significance as to the locality of disease. It may be excited by contact when it is not spontaneous, and probably represents the highest degree of excess of impressions conveyed by the nerves of tactile sensibility. The still vaguer feeling called "numbness" is also common, and may occur, in slight degree, in diseases of which all other symptoms are purely motor. But this word is used in so many different senses that the meaning in which the individual uses it should always be ascertained as far as possible. Sometimes it signifies distinct loss of sensibility. More often a feeling as if there ought to be loss when there is not. Our sensations altogether transcend our vocabulary, but the observer must try (without leading) to ascertain the character of the sensation experienced.

REFLEX ACTION.—*Loss of reflex action* indicates an interruption of the reflex arc concerned. This interruption may be anywhere between the peripheral endings of the motor and sensory nerves, and thus is not necessarily within the spinal cord, or even within the spinal canal. It is as constant in peripheral neuritis as in any spinal disease. The position of the interruption must be determined by the associated symptoms; if it is in the centripetal portion of the reflex arc, there is impairment of sensation, since the interruption will equally arrest conduction to the brain. If it is in the centrifugal portion of the arc, there is a corresponding interruption in the path of the voluntary impulse, and loss of motor power. Moreover, disease of the motor centre or motor nerves causes also degeneration of the nerves, and wasting of the muscles. Any considerable disease of the nerves abolishes al reflex action from the part they supply, but disease limited to th motor structures may permit a reflex movement to take place at distance, although preventing it at the part stimulated.

Disease of the motor centre or nerves causes loss of all form of reflex action, the simple form and the muscle-reflex that underlie myotatic irritability. Partial disease of the afferent path may impair one and not the other, may abolish the muscle-reflex action (myotatic irritability) and not the superficial reflex action, since the afferent nerves for the two are distinct, coming in the one case from the muscles, in the other from the skin. When one only is lost it is generally the muscle-reflex action; the nerves for this seem more susceptible and to have less power of resisting morbid influences.

Loss of all reflex action may occur as a transient symptom, immediately after the onset of an acute lesion of the cord, apparently from irritative inhibition of the centres. Cutaneous reflex action may be lessened permanently in some cases of brain disease on the side of the motor palsy, even when the muscle-reflex action is increased. Indeed, this opposite change in the two forms of reflex action co-existing on the same side always suggests intra-cerebral disease. It may be well again to remind the reader how difficult it often is to be sure whether the knee-jerk is present or is lost, on account of the readiness with which its occurrence may be prevented by inability to relax the muscles, and its presence may be simulated by a true reflex action (see p. 200).

Excess of reflex action implies, of necessity, the integrity of the reflex arc concerned, and shows that organic disease, if it exists, is higher up the cord. Each form of reflex action is often increased. In some acute diseases, as acute meningitis, and probably also in some chronic diseases, the increase may be due to an irritation of the centres, but in most forms of chronic disease it is apparently the result of a loss of control, and indicates disease between the centre concerned and the brain. We have already considered (p. 202) the probable mechanism, and have seen that the excess of the muscle-reflex action is related to disease of the pyramidal fibres, and especially to the loss of the terminal part of these fibres, within the grey matter, adjacent to the centres concerned.* This is the significance of considerable excess. Degeneration of the terminal portions of the fibres is commonly due to a descending degeneration of the fibres themselves. It is possible, however, that the degeneration of the endings of the pyramidal fibres may be primary, as is that of the nerve-fibres. (See Primary Spastic Paraplegia.) The increase of reflex action is chiefly manifested in the legs, in which reflex action is normally more active and important than in the arms. The increase in the cutaneous reflex action is shown by its occurrence on a slighter stimulation of the skin, and by a greater degree and extent of the muscular contractions produced. In these the flexors usually predominate, so that the leg is drawn up in a strong movement. As the action of the sexual organs depends, in part, on superficial reflex processes, priapism is common when the reflex excess is great. The increase of the muscle-reflex action is shown by an increase of myotatic irritability, and therefore of the so-called "tendon-reflex contractions,"—the increased knee-jerk, rectus-clonus, foot-clonus, wrist-jerk, elbow-jerk, chin-jerk (jaw-jerk), &c. These have been already described. As already stated, the excess of this irritability is at first moderate, and slowly increases. A high degree of over-action is never suddenly attained, as it may be in the case of cutaneous reflex action. It would seem as though, in the centres liberated from control, a capacity for excessive action is slowly developed, which ultimately results in tonic spasm.

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* It is interesting to note the analogy between the effects of degeneration of the termination of the two segments of the motor path. The increased activity of the muscle-reflex centres, which results from degeneration of the termination of the upper segment, presents some similarity to the increased voltaic irritability of the muscular fibres which results from degeneration of the termination of the lower segment.

Not only is the common cause of this excess damage to the pyramidal tracts higher up the cord, entailing their secondary degeneration, but if such disease higher up—as, for instance, primary myelitis or injury to the cord—is followed by abolition of the muscle-reflex action in the parts below, it is the result of inflammatory damage to the lower part of the cord in which the centres are situated, generally by a descending myelitis. Sometimes the original myelitis involved the whole or a considerable part of the cord below, and the indications of a transverse lesion were merely those of the upper limit of the disease. It is not uncommon for the secondary myelitis below an original transverse lesion to succeed it, and abolish the reflex action which was at first unimpaired. I have several times met with such a sequence. A primary dorsal myelitis has left reflex action in the legs unimpaired and tending to excess, with the sphincter ani in its condition of uncontrolled automatic action, when after a few days, a week or more, indications of a recurrence of myelitis have been attended by abolition of all reflex action, including the knee-jerk, by flaccidity of the muscles, with rapid development of the reaction of degeneration in them, and by complete relaxation of the sphincter ani. This is the state from the first in the cases in which the primary myelitis involves the whole lower part of the cord. Sometimes, indeed, such descending inflammation is limited to the grey matter or to the posterior columns, with corresponding limitation of the symptoms but with a similar abolition of reflex action. Of course, such descending inflammation is a very different thing from the secondary descending degeneration of the pyramidal tracts.*

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* In consequence of the occurrence of this inflammation in the lower part of the cord having been overlooked, it has been thought that a transverse lesion higher up has the power of abolishing reflex action below, where the cord is not damaged except by descending degeneration. See, however, p. 229, note.

Over-action may perhaps be also produced by a state of irritation propagated from above, as in the "early rigidity" of hemiplegia; we have seen that the motor impulses probably pass from the pyramidal fibres through part at least of the muscle-reflex mechanism. Primary over-action seems also to occur, although apparently it only attains a slight degree. It is probably the cause of the trifling excess met with in cases of defective nutrition of the spinal cord or of the nervous system generally.

Reflex Spasm.—We have seen (p. 16) that the muscular state on which myotatic irritability depends, assumed to be due to a muscle-reflex action, is probably identical with physiological *tone*. Whenever there is a considerable increase in the irritability, there is a tendency to tonic spasm, in which there is a balanced contraction of the muscles, fixing the limbs in the position of extension. At first there is merely slight stiffness of the legs, especially felt on waking from sleep; afterwards the legs, when extended, become distinctly stiff, although they may be quite supple when flexed. If gradually extended, when near full extension, the spasm suddenly comes on and completes the movement, as the blade of a pocket knife moves under the influence of the spring. Hence this has been termed "clasp-knife rigidity." Ultimately the extensor spasm may be so great that, when it occurs, both legs are rigid, and are so firmly connected with the pelvis that if one is lifted from the bed the other is moved also. This is only met with in organic disease, and is an important diagnostic indication. Paroxysms of spasm may be brought on by any impression on the sensory nerves, a prick on the skin, or an attempt to obtain the clonus. The extreme spasm prevents any clonus being obtained, and it may be necessary to flex the limb before an attempt to obtain it is successful. As violent spasm is passing off, moreover, the tension of the muscles may alone set up a clonus, so that the paroxysm of tonic spasm passes into clonic spasm, a phenomenon that was termed by Brown-Séquard "spinal epilepsy," on account of the superficial resemblance to the sequence of spasm in an epileptic fit. This clonic spasm may often be arrested by any strong painful impression on the sensory nerves, a pinch of the skin, or painful flexion of the big toe; the strong stimulation of the nerves of common sensation inhibits the reflex centre concerned in the clonus. There is always much weakness of the legs when there is much spasm, and there may be almost complete paralysis. Hence the condition has been termed "spastic paraplegia." It is probable that the central reflex mechanisms concerned in the production of this extensor spasm are those that subserve the act of standing. The spasm often enables a paraplegic patient to stand, when his voluntary power is quite insufficient for the act. In advanced cases the extensor spasm may be varied by attacks of flexor spasm, which at first come on chiefly during sleep, but after a time preponderate, and may ultimately be permanent; this is always an unfavorable symptom, since, for some reason not yet understood, this flexor spasm indicates a condition of the spinal cord from which recovery is rare.

VASO-MOTOR AND TROPHIC DISTURBANCE.—*Changes in Nutrition.*—Considerable wasting of the *muscles* indicates disease of the motor nerve-cells, or of the nerve-fibres proceeding from these cells. If the disease is acute, there is rapid degeneration of the nerves, with the reaction of degeneration in the muscles (see p. 56). The wasting in these cases is always great, and there is always loss of reflex action. Occasionally some fibres of the nerve and muscle suffer and others do not, causing the "middle" or "mixed" form of reaction (see p. 26). In rare cases, in which the nerve degeneration set up is of an intensely irritative character, the muscles may rapidly lose all irritability in consequence of true degeneration of the fibres. I have observed this, for instance, in a growth in the lumbar region. In other cases of disease, however, there is a slighter degree of wasting of the muscles; there is no loss of irritability in the nerves; the irritability may be slightly increased or lessened, but is changed in the same way to each form of electricity. In this condition reflex action is not lost, but, on the contrary, is often increased. The change is apparently the result of a peculiar alteration in the nutrition of the nerve-cells. It is usually the effect of an *irritative* degeneration of the pyramidal fibres, which influences in a peculiar manner the nutrition of the motor cells.

The changes in the nutrition of the skin, if slight and chronic, resemble those produced by disease of the nerves (see p. 61), and are probably produced through the agency of the posterior roots. Sometimes, however, these changes are most acute. Very slight pressure, continued for a few hours, causes vesication, and even sloughing of the skin. Sometimes bullæ form where there has been no pressure. Occasionally there is inflammation of the joints. Such intense changes are only met with when the morbid process in the cord is irritative in its character, especially in hæmorrhage, and in spreading or disseminated myelitis. The points on which sloughing occurs with greatest readiness are the heel, over the malleoli, the trochanters, and the sacrum. The position of the bedsores in paraplegia, usually over the sacrum, is different from that in hemiplegia, generally over the gluteal region on the paralysed side. The tendency to trophic disturbance probably favours the occurrence of cystitis from retention of urine, and the readiness with which the inflammation invades the kidneys. I have seen a low form of suppurating subperitoneal pelvic cellulitis, in a case of disseminated myelitis with cutaneous sloughs, in which no other cause for the cellulitis could be found.

Vaso-motor disturbance is common in slight degree, irrespective of the seat of the disease, but, like the trophic changes, which probably depend, in some degree, upon it, it is always most intense in irritative lesions of the cord. The slighter and common degree consists merely in increased warmth of the limbs in which the vessels dilate too readily, while, at a later period, the limbs are colder, paler, and often livid at the extremities. In rare cases intense flushing of the skin has been observed, or copious and long-continued perspiration. When the disease is in the lower cervical region, flushing and perspiration may occur on the face and head.

Disease of the cervical cord or nerves is sometimes attended by a change in the pupil, on one side only. It is due to the fact that the dilator is innervated from the cervical sympathetic through the last cervical nerves. There may be contraction, from paralysis of these radiating fibres, or there may be persistent dilatation from their irritation. The cause of the double myosis, so common in some degenerative diseases of the cord, is uncertain; it is associated with loss of the light-reflex, and also of the reflex dilatation produced by stimulating the skin of the neck.

The frequency of the heart's action is often permanently increased in locomotor ataxy, but we do not know by what mechanism. In disease of the cervical cord (perhaps also in that of the cervical nerve-roots) the heart may be slowed to forty, thirty, or twenty beats per minute, usually only for a time. The effect may perhaps be due to a loss of the influence of the nerves which, when stimulated, quicken the heart. In acute disease of the upper dorsal region the heart's action may be persistently frequent.

Visceral Disturbance.—Various disturbances in the *alimentary canal* occur in disease of the spinal cord, and are no doubt due to the fact that the sympathetic nerves immediately controlling the canal are under the influence of the cord, and may be excited to abnormal action by its irritation, or may underact when its influence is lessened. Difficulty of deglutition is observed in some cases of acute disease of the upper cervical region. Vomiting occurs also in disease of the same part, especially in fracture of the cervical vertebræ. Constipation is extremely common in disease of the cord above the lumbar enlargement.

Paralysis of the Sphincters.—Incontinence of urine and fæces may be due to disease of the centres that control the action of the sphincter ani and the muscular mechanism of the bladder respectively, or it may be due merely to disease of the path (probably the pyramidal tract) through which voluntary control is exerted. In the latter case the sphincter ani acts in an automatic reflex manner as already explained. If the finger is introduced within it, the initial relaxation is felt to be succeeded by a firm tonic contraction. This proves that the anal centre in the lumbar enlargement, and the nerves proceeding from it, are intact. If this centre is destroyed, or the nerves interrupted, no tonic contraction can be felt; there is complete and persistent relaxation. I have more than once observed a dorsal transverse myelitis to arrest the voluntary influence and leave the sphincter in its condition of simple reflex action, and, a week or ten days later, the extension of the myelitis into the lumbar region to be attended by (among other signs) perfect inaction of the sphincter. A rectal examination will then often give important information regarding the extent of the disease downwards.

The sphincter of the bladder is not accessible to such direct examination, and the mechanism seems to be more complex, but we may distinguish conditions corresponding to those of the rectum. If the lumbar centre is destroyed the sphincter is permanently relaxed; urine dribbles out of the bladder as fast as it enters it; there is simple incontinence. If there is an interruption of the voluntary path above the centre, the sphincter may act automatically; when a certain amount of urine has collected in the bladder it excites the muscular fibres in the wall to contract; the sphincter relaxes, and the urine escapes; there is intermittent incontinence. In this condition the mechanism is excited to action by any pressure on the bladder; a movement, or the act of coughing, will make the urine come away. But the muscle of the wall of the bladder is indirectly under the influence of the will; when the sphincter is voluntarily relaxed the detrusor fibres in the wall contract. Loss of voluntary power seems to lead to weakness in the wall of the bladder, so that the bladder is never perfectly emptied, and ultimately the detrusor cannot contract to expel the urine, even when the sphincter is relaxed. Hence retention of urine occurs. When a certain degree of distension of the bladder is attained, the pressure becomes sufficient to force the urine out whenever the sphincter relaxes, and such relaxation is continually occurring under the high pressure that is repeatedly renewed in consequence of the flow from the kidneys. Hence incontinence results—"overflow incontinence." The retention often develops very gradually and insidiously when the voluntary power is only lessened; the residual urine is at first small in amount, but gradually increases as the detrusor fails to contract adequately against the resistance. It is important, if the urine comes away involuntarily, to ascertain which form of incontinence exists; since overflow incontinence, and the retention it indicates, have much graver consequences than simple incontinence. The permanent distension of the bladder constitutes a permanent hindrance to the flow of urine from the kidneys, and serious kidney disease may result. This is a not uncommon cause of death in spinal disease. Moreover, whenever the bladder is habitually emptied imperfectly, decomposition is apt to occur in the residual urine. The decomposition is often aided by the lessened acidity, or even actual alkalinity, of the urine. This change in the composition of the urine excites inflammation of the bladder; pus is formed by the mucous membrane, and this, in its turn, increases the decomposition of the urine. The damage to the kidneys, by the hindrance to the flow of urine from them, may be increased by inflammation ascending the ureters from the bladder.

Pyrexia.—The initial elevation of temperature in some spinal lesions may be due to them directly, but sometimes it is due to a blood-state of which the cord disease is a result. This is probably the case in polio-myelitis. The temperature is sometimes raised in disease of the cord apart from the influence of morbid process. Disease of the upper cervical cord, like that of the medulla, may cause hyperpyrexia, and this may also result from acute central lesions that occupy a considerable vertical extent, such as a central hæmorrhage, although by what mechanism these act we do not know. Slight variable elevation of temperature sometimes occurs in disease of the dorsal cord, and is probably due, in some way, to the disturbance of its functions rather than to any influence of the morbid process. For instance, in one case of hæmorrhagic myelitis with damage from the third dorsal to the second lumbar segments, every application of electricity to the legs was followed by slight pyrexia.

Convulsions.—Lastly, convulsive attacks have been known to attend the onset of acute lesions in various parts of the cord, in adults as well as in children. They are most frequent in disease of the cervical cord, but I have known a convulsion to occur in an adult at the onset of myelitis in the lower dorsal region. The convulsions are general. Their mechanism is unknown, and in some cases, especially of infantile polio-myelitis, they may, like the mitral pyrexia, be the result of general disturbance of the system by an acute morbid blood-state.

COMBINATION OF SYMPTOMS.—Certain symptoms are frequently combined, and their combination has a definite localising significance. Loss of motor power occurs in two different associations, according to the position of its cause in the motor path. It may be combined with muscular wasting, loss of electric irritability, and loss of all reflex action. In this case the disease is in the anterior cornua or nerve-roots, the lower segment of the motor path. (But the same symptoms are produced by disease of the nerves themselves—in multiple neuritis.) On the other hand, there may be no wasting, no change in irritability, and, instead of a loss of reflex action, the myotatic irritability is increased. The interruption of the motor path is then in the upper segment, usually in the pyramidal tracts. It may be a focal lesion of the cord, or a primary degeneration of the tracts. The distinction is that, in the latter case, there is nothing more than the muscular weakness and increased myotatic irritability. In the former case there are, or have been, indications that the lesion has extended beyond the purely motor elements.

The symptoms caused by disease of the several elements of the cord have now been described, but it is necessary to consider further the combination of symptoms that results from a total transverse lesion of the cord, and from a unilateral lesion.

A *total transverse lesion*, however limited in vertical extent, separates from the brain all parts below it, and hence, so far as will and feeling are concerned, it produces the same effect as if the whole of the cord below the lesion were destroyed. Compression of the cord in the middle of the cervical enlargement, for instance, abolishes motion and sensation in all parts below the distribution of the cervical plexus. A transverse lesion at the second dorsal nerve causes motor and sensory palsy of the trunk and legs, leaving the arms unaffected. But the same loss, motion and sensory, results from disease which damages the whole cord up to the level indicated. Hence the extent of the motor and sensory paralysis indicates only the upward limit of the lesion; how far it extends downwards is shown by the interference with the central and especially the reflex functions of the cord. The upward level is indicated not only by the loss of conduction, but also by the position of the girdle-pain, and radiating pains, or zone of hyperæsthesia, which are due to the irritation of the lowest sensory nerves in the upper portion.

It is desirable to know the symptoms of impaired conduction that occur when a transverse lesion is at different levels in the spinal cord. They may readily be ascertained by an examination of the table given at p. 210. There is, however, some uncertainty as to the effect on sensation in the limbs produced by disease at different parts of the lumbar and cervical enlargements, since, as we have seen, the exact representation of sensation in the cord is still uncertain.

The upper limit of the lesion is shown by the upward extent of the motor and sensory loss, according to the first three columns. The lowest nerves supply the anus and perinæum. Those that supply the skin and muscles of the leg and foot arise from the fourth lumbar to the third sacral segments, and are damaged by a lesion involving the lower part of the lumbar enlargement. We must, however, remember that the skin of the inner part of the leg is not supplied from the sacral nerves, and so may escape when the outer part of the leg and back of the thigh have lost sensation. In the middle of the lumbar enlargement the nerves arise which enter the lumbo-sacral cord, and these are probably destined for the flexors of the knee, and for the hip-muscles supplied by the sacral plexus, the glutei, quadratus, and gemelli, and for the skin of the lower part of the gluteal region. These parts then will be paralysed by disease in the middle of the lumbar enlargement, while the muscles and skin in front of the thigh are unaffected. The latter suffer when the disease affects the upper part of the lumbar enlargement, the origin of the anterior crural (rectus, &c.), and obturator (adductors). The skin on the upper and outer parts of the thigh loses sensibility, with the part adjacent to the scrotum, and in the groin, only when the disease damages the highest part of the lumbar enlargement, from which the second and third lumbar nerves arise, and then the flexors of the hip become paralysed. In proportion as the disease is higher in the dorsal region, we have the symptoms ascending higher up the trunk, and marking accurately the height of the lesion by the loss of cutaneous sensibility, and by the impairment—first, of the abdominal muscles, and then of the intercostal muscles. The umbilicus corresponds to the tenth dorsal nerves, and the ensiform area to the sixth and seventh. When the disease reaches the lowest part of the cervical enlargement (the first dorsal nerves), we have the first symptoms in the upper extremity; but these are not, as might be expected, in the muscles moving the shoulder-joint, but in the hand. The first numbness is complained of in the little finger, and the first weakness is in the intrinsic muscles of the hand. Ascending higher, the symptoms pass up the arm with some uniformity, and without respect to nerve distribution. When the middle of the cervical enlargement is reached (the fifth, sixth, seventh cervical) the shoulder-muscles and the serratus magnus become paralysed, and there is general loss of power and sensation. (For details see table, p. 210, and also p. 192.) Above the level of the sixth pair, the trapezius and sterno-mastoid become somewhat weakened, for the fibres of the spinal accessory which supply them undoubtedly arise in part from this region of the cord. At the fourth and fifth cervical the lower part of the neck becomes anæsthetic, and the diaphragm ceases to act. Here our localisation might cease, for total transverse lesions at this spot necessarily cause death. For a little time the sterno-mastoids and scaleni can still get some air into the chest, rarely in sufficient amount to maintain life for more than a few days. But limited lesions may occur higher up, and then we have complete powerlessness of the muscles moving the head, upper part of trapezius and sterno-mastoid, and other muscles attached to the occipital bone, and interference with sensation in the neck and parts of the head, which are not supplied by the fifth nerve.

The extent downwards of the lesion, its vertical extent, is thus not indicated by the impairment of the conducting functions, by the motor or sensory paralysis; to ascertain it we have to examine the functions of the cord as a central organ, and to ascertain how far they are impaired in the paralysed region—to examine especially muscular nutrition and reflex action. The state of muscular nutrition and irritability indicates how far the anterior cornua are injured. The relation of the several groups of muscles to the cord is shown in the first column of the table. The integrity of reflex action indicates the integrity of the reflex loops, and the study of the superficial reflexes of the trunk is especially instructive in this respect. The series of reflexes, and the relation of each to the cord, are shown in the third column of the table; the muscle-reflex contractions are printed in italics in the position which corresponds to the centres and nerve-roots which are essential for their production. Excess of superficial reflex action indicates loss of the cerebral control exerted on the reflex centres, and considerable excess of the muscle-reflexes implies impaired function of the lowest part of the pyramidal tracts, and generally shows the existence of a descending degeneration in the lateral columns. Reflex action in the legs may be abolished for a few hours by acute disease or injury above the lumbar enlargement, in consequence of inhibitory shock, but soon returns if the lumbar centres are intact. As already explained (p. 220), if there is no return of reflex action or myotatic irritability, their absence shows that the lumbar region is involved in the primary lesion; secondary extension to this is indicated by loss of reflex action distinctly subsequent to the onset. If such loss occurs without wasting and loss of faradic irritability in the muscles it shows that the disease in the lumbar region is limited to the posterior columns or cornua.*

The fact of chief importance is that dorsal cord disease, the most common form, causes no persistent loss of reflex action in the legs, and that for such loss special causes must be sought.

Unilateral lesions interrupt the motor path to the muscles on the same side as the lesion, causing one-sided palsy, termed "spinal hemiplegia" when the disease is so high as to affect both arm and leg, "hemi-paraplegia" when it is lower, and affects one leg only. There is often some loss of power on the opposite side, which may be due either to slighter damage to the other side of the cord (since few lesions are strictly unilateral) or to damage to non-decussating or re-decussating fibres. Conversely, the paralysis of the leg may be incomplete when that of the arm is complete, owing to the escape of the fibres for the leg which cross lower down the cord. Sensation is affected on the opposite side, but not quite up to the level of the lesion, because the decussation of the sensory tract is not immediate, but occurs somewhat above the entrance of the nerves. The upper level may vary for different forms of sensibility, in consequence probably of the level of crossing (in relation to entrance) being different for the several paths. A lesion in one side of the lumbar enlargement often affects sensation on the same side as motion, because it damages the sensory path before it has crossed. In all cases of crossed motor and sensory paralysis, the sensibility of the muscles differs from the other forms of sensibility, and if it is affected on one side, this is the side of the motor palsy and not of the cutaneous anæsthesia.†

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* A few cases have been met with in which a transverse lesion of the dorsal cord, especially a transverse concussion-myelitis, without post-mortem signs of lumbar inflammation, has caused a persistent loss of reflex action in the legs. In these cases, however, there has been not only loss of the muscle-reflex action, but also of the skin reflexes, a clear indication of an exceptional condition. Moreover, there has also been loss of faradic irritability and rapid muscular wasting—in short, there have been all the indications of a lumbar myelitis. The most probable explanation of these cases is that the descending degeneration of the pyramidal tract has been more than usually irritative in nature, so as practically to amount to a parenchymatous inflammation, and that this has invaded the motor structures as a nutritional change, sufficient to abolish their function, without, however, destroying their form. In one such case it was noted that the lumbar nerve elements were extremely granular in aspect. For a different explanation of these cases, see Bastian, 'Med.-Chir. Trans.,' 1890. also 91. 552

† This was first pointed out by Brown-Séquard in his important study of these cases. Scepticism has been expressed as to the fact, based on a denial that the test commonly employed, recognition of posture, is significant. Experiments on animals, even monkeys, are inconclusive, but the evidence from cases in man is very

The crossed affection of cutaneous sensibility may involve all forms of sensation or only some of them. Sensibility to pain is almost invariably impaired. The temperature sense is usually affected with that for pain; in only two of twenty recorded cases (in which the affection of sensibility was carefully noted) was the sense of temperature normal, and that of pain impaired, and in neither of these cases was the sensibility to pain actually lost. On the other hand, in one third of the cases tactile sensibility was unaffected, and in about one tenth of the cases it was impaired on both sides.

Cutaneous sensibility is sometimes impaired in a zone at the level of the lesion, and on the same side, in consequence of the damage to the nerve-roots entering the cord, and above this there may be a narrow band of hyperæsthesia from irritation of the roots at the upper part of the lesion. On the side of the lesion, below the anæsthetic zone, there is, in most cases, a remarkable hyperæsthesia, corresponding in distribution with the anæsthesia on the opposite side. Painful impressions are felt most acutely, and even a touch on the skin, or warm or cold bodies, produce pain. The cause of this hyperæsthesia is obscure. Both it and the opposite loss of sensibility may last for twenty years, and it cannot be ascribed therefore to any irritation by the morbid process. It is perhaps due to an altered action of the cerebral centres on the opposite side of the brain. There is much evidence of an intimate connection between the sensory centres on the two sides, and it is conceivable that the altered functional state of the centre on the side of the lesion, to which impressions cease to come, may induce in the opposite hemisphere a condition expressed as hyperæsthesia. The condition may be thought of in relation to the peculiar phenomena of transfer in hysterical hemianæsthesia.

Reflex action is increased in all its forms on the side of the lesion, but the increase only occurs after some days. At first it is lessened or even abolished, no doubt from the inhibitory influence of the irritation of the morbid process.

The side below the lesion is at first, for some days or even weeks, warmer than the other, the difference being usually about a degree Fahrenheit. In the course of time this difference passes away, and the side may even be colder than the other.

These symptoms may be shown in the form of a table:

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strong, not only of the occurrence of the loss, but of the significance of the test. In some cases, moreover, the muscles were insensitive to pressure. One recorded instance is the following:—A stab in the dorsal region caused loss of cutaneous sensibility in the right leg and motor palsy in the left. On this left side, on which cutaneous sensibility was normal, the sense of posture was absolutely lost, while it was present on the right side on which cutaneous sensibility was impaired. The patient often thought the left leg was flexed when it was extended. Ultimately this leg presented distinct ataxy. (Gilbert, 'Arch. de Neurologie,' 1882, p. 275.)

Zone of cut. hyperæsthesia. ——— anæsthesia.	LESION.	
Motor palsy. Hyperæsthesia of skin. Muscular sense impaired. Reflex action first lessened, then increased. Temperature raised.		Muscular power normal. Loss of sensibility of skin. Muscular sense normal. Reflex action normal. Temperature same as that above lesion.

The vertical distribution of the symptoms varies according to the vertical position of the lesion. Their relations are best marked when the lesion is in the dorsal region of the spinal cord. When it is in the cervical or lumbar enlargement the upper limit is less distinct because of the complex representation of skin and muscle in the nerve-roots.

GENERAL PATHOLOGY AND ETIOLOGY: INDICATIONS OF THE NATURE OF THE LESION: PATHOLOGICAL DIAGNOSIS.

The kinds of disease to which the spinal cord is liable are not specifically numerous, but those which are common present several varieties. The processes are congestion and anæmia, hæmorrhage, inflammation, degeneration, and growths. Hæmorrhage and growths are, on the whole, rare. Congestion and anæmia play an uncertain part in the production of symptoms; their influence has been certainly overrated by some writers, and perhaps under-estimated by others. The most frequent lesions are inflammation and degeneration. These two processes, distinct in their typical forms, blend in their intermediate varieties. Inflammation varies much in character and course. Its effects are, in some cases, produced with great rapidity, in other cases with extreme slowness. It probably commences in most instances outside the nerve-elements, which are secondarily involved. Perhaps some acute processes in the nerve-elements themselves are most accurately regarded as a parenchymatous inflammation, analogous to that which we know occurs in the fibres of the peripheral nerves. Degeneration consists of two processes, a wasting of the nerve-structures, cells and fibres, and an overgrowth of the connective-tissue elements. The latter leads to the condition termed sclerosis. The name has been given to it, apparently, because the process is one that, in other organs, increases their consistence. In the cord it has not often this effect. The newly-formed connective tissue is rarely of greater firmness, and is often less firm, than the nerve-elements it replaces. In sclerosis, however, we must distinguish two types. In one the process is limited to structures that have the same function; the process begins as a degeneration of the nerve-elements, and the overgrowth of the connective tissue is a consequence of their wasting, the affection is essentially "parenchymatous." The so-called "secondary degenerations" are of this character; but similar changes are often primary, and affect the structures that have the same function, often through a considerable extent of the cord. Hence they are termed "system diseases." In the other type the morbid changes are not distributed according to function. They are apparently random in incidence, and involve adjacent structures in consequence of contiguity. These begin in the connective tissue and not in the nerve-elements, which suffer secondarily. They are essentially interstitial processes. Insular sclerosis is an example of this type.

The position of the islets of sclerosis has no relation to the function of the parts, and may involve parts of adjacent structures that have no common function. Areas of diffuse sclerosis are also met with which have not the sharp limitation of the insular form, and are regarded by some as of the nature of chronic sclerotic inflammation. If such a lesion is so situated as to involve fibres that undergo secondary degeneration, this necessarily results when the damage to the fibres is sufficiently great. We have then a combination of the random and systemic forms of sclerosis, but one that has no real pathological significance.

There are, however, other relations between the two types that deserve attention. These will be considered in connection with the special morbid processes that present them, but a brief mention of them here may make some facts of other diseases more intelligible. The process of secondary degeneration and secondary sclerosis varies in its character, presenting in some cases a greater amount of vascular disturbance than in others. It seems to partake of the character of the lesion causing it, as we have seen (p. 55) the process of secondary degeneration in the nerves may do. The same difference is seen in the primary degenerations, such as occur in tabes. This difference is analogous to that which, as already described, occurs in some varieties of multiple neuritis. We can indeed see the difference in the eye: tabetic optic nerve atrophy, in most cases, presents no sign of inflammation, while in others the early stage may be accompanied by distinct slight neuritis, to be seen with the ophthalmoscope. Secondly, the chronic inflammations, with the diffuse limitation, extending to adjacent structures irrespective of function, have yet sometimes a marked tendency to be localised in structures of definite function. Thus there is sometimes diffuse sclerosis of the lateral pyramidal tracts and posterior median columns, not sharply limited to these, and yet with a correspondence and symmetry not to be overlooked. Thus the two processes tend to meet; the systemic degeneration or sclerosis to be diffuse; the diffuse inflammation or sclerosis to be systemic; and it is sometimes not easy to say, from mere microscopic examination, to which class a lesion belongs. Lastly, there is a curious fact, which has been strangely overlooked although attention was first called to it by Charcot—that inflammation may extend from a primary lesion along a tract that undergoes secondary degeneration, but in the opposite direction to that of the degeneration. It is very common for the inflammation at a given place in the cord, especially that due to compression, to extend farthest along certain tracts, irrespective of their secondary degeneration, and in some cases this extension occurs through a considerable extent of the cord. An ascending inflammation limited to the pyramidal tracts may, for instance, be traced through several segments. The fact suggests that the neuroglial elements may share to some extent a difference that obtains between the fibres of different tracts, and it may easily lead, and probably has led, to erroneous conclusions.

Softening of the spinal cord is very common. Whenever the nerve-fibres are broken up into disconnected globules of myelin, separated by serum in a sort of emulsion, the consistence of the part is necessarily lessened. Acute inflammation always causes such a breaking up of the nerve-fibres, and hence inflammation always causes softening as its first effect. The separate particles are augmented by leucocytes, which escape abundantly into the tissue. Ultimately, connective-tissue elements are formed, which increase the consistence, it may be up to, and even beyond, the normal degree. In a very chronic inflammation the formation of new tissue may proceed *pari passu* with the destructive process, and there may be at no time much diminution of consistence. It is in these cases that it is difficult to draw the line between inflammation and degeneration.

Does such softening of the cord occur apart from inflammation? The question is not easy to answer. In the brain, necrotic softening is very common as a consequence of arterial obstruction—is indeed the common form of softening. But in the spinal cord we have no evidence of the occurrence of necrotic softening. If embolism occurs, it is excessively rare. The course of the vessels does not favour the passage of a plug into them, while the vertical connection in the anterior spinal and anastomotic arteries (see p. 188) will prevent damage from obstruction in the central system unless this is situated in the terminal vessels of the grey matter. Arterial thrombosis, due to atheroma of the walls of the vessels, such as is so common in the brain, probably does not occur in the cord. The arteries are smaller than those in which atheroma is met with in the brain, and the lowness of the blood-pressure within them involves the absence of the chief cause of atheroma. It is possible that spontaneous thrombosis may sometimes set up the changes that are now regarded as those of primary inflammation; and such a lesion has actually been met with, but only in isolated form, and we have no evidence of its frequency.

Besides the morbid processes to which the cord itself is liable, it suffers also in consequence of disease outside it. It may be compressed by growths springing from the membranes or bones, and by products of inflammation within the spinal canal. Compression not only causes degeneration of the nerve-elements, but usually excites actual inflammation. This "compression-myelitis" may attain a degree and an acuteness out of all proportion to the causal compression. The membranes may be the seat of hæmorrhage which compresses the cord, or inflammation which irritates it, even when the organ is not invaded in appreciable degree.

The series of recognised lesions of the spinal cord, obvious or microscopic, does not by any means exhaust the list of its morbid states. Changes may occur in the nutrition of its elements interfering with their function, which are, and are likely to remain, altogether beyond our means of detection. Such morbid states are often called "functional diseases," but when prolonged they are probably more accurately conceived as nutritional diseases (see p. 1). Derangements of function that we can regard as purely such are few and rare. But disturbances of function may be due to organic disease in some other part of the nervous system, and they may also be the result of toxic agents.

Lastly, functional action in all organs is attended with increased blood-supply; and when excessive, the vascular disturbance may go on to a condition indistinguishable from inflammation, with escape of leucocytes, &c. The prolonged experimental stimulation of sensory nerves has actually produced myelitis in the related portion of the cord.

In connection with the general pathology of diseases of the spinal cord, it is important to note certain general facts of their causation:—(1) *Neuropathic disposition.*—An inherited tendency to disease of the nerve-elements, manifested by such affections as epilepsy and insanity. This cause is chiefly influential in producing the structural and nutritional diseases, beginning in the nerve-elements, and especially, among structural diseases, the "system-degenerations," as they are called. (2) *Prolonged mental distress* sometimes sets up degenerative changes in some elements of the cord, especially in persons who are predisposed by (1). (3) *Injury.*—A severe concussion of the cord may cause (*a*) instant grave damage, usually hæmorrhage. Or (*b*) it may cause no immediate effect, but symptoms may come on at the end of a few days and progress slowly or rapidly. Such symptoms are usually due to inflammation, secondary to minute spots of injury, too small to cause direct symptoms. Lastly (*c*), the concussion sometimes seems to pervert the process of nutrition in the nerve-elements. Slow symptoms of impaired function may result, and these may progress, until, after months or years, there is actual structural disease. (4) *Exposure to cold.*—Wet cold is especially effective. The most frequent effect of a severe exposure is acute inflammation, but habitual exposure may produce chronic inflammation, or degenerative disease. The exposure that is effective may be general, or chiefly of the feet and legs, occasionally of the back. (5) *Toxic agents* that can act on the cord are numerous and are seen in alcoholism and lathyrism. (6) *Other diseases* may also induce morbid changes in the spinal cord. The primary malady may be an acute specific disease, or some more obscure blood-state, the precise nature and relations of which are still undecided. It is probable that certain blood-states, due to an organised virus, excite some forms of inflammation of the cord and of its membranes, just as it may cause inflammation of the peripheral nerves. Septicæmia may excite meningitis, or even abscess of the cord itself. Tubercle is common on the membranes, but seldom produces inflammation. Diphtheria may cause acute changes in the nerve-cells and nerve-roots. Syphilis is a very frequent cause of disease, and may act in several ways. The demonstrated mechanisms are (*a*) by a syphilitic growth, compressing or invading the cord, and (*b*) by chronic syphilitic meningitis, damaging the cord and the nerve-roots. But (*c*) acute and chronic inflammations of the cord are often met with in syphilitic subjects, and have been thought to be, in many instances, of syphilitic origin. It is doubtful whether they present any syphilitic characters, *i. e.* any histological features by which they differ from non-syphilitic inflammations. The evidence of dependence on syphilis is stronger in the case of chronic and subacute disseminated inflammation than in the case of acute myelitis. Lastly (*d*), certain degenerative diseases of the cord are very commonly preceded by syphilis; one of them, locomotor ataxy, so frequently that a causal relation between the two can scarcely be doubted. But these degenerative diseases are certainly not syphilitic in nature; they differ in no respect from the similar morbid processes that occur in individuals who have not had syphilis, and are probably the consequence of some virus left behind by the morbid agency to which the constitutional malady is due.

The outline just given of the general pathology and etiology of diseases of the spinal cord will enable us to consider the last element in diagnosis, the nature of the lesion. The seat of the disease is indicated by the combination of symptoms; its nature can only be determined by considering, separately and together, several other points:—(1) The way in which the symptoms came on. (2) The causes that can be traced, taken in conjunction with the known effects of those causes. (3) The seat of the disease, taken in conjunction with the known liability of certain structures to certain lesions. The most important of these elements is the mode of onset, and the other indications should only be used in strict subordination to this.

The time occupied by the onset of the disease is thus the first element in the pathological diagnosis. By "time of onset" is meant the period that elapses between the actual commencement of the symptoms and their attainment of a considerable degree of intensity. We may divide the chief modes of onset into five classes, and classify the most common lesions in relation to them, in the following table:

DISEASE.	ONSET.	DISEASE.
	Sudden (few minutes)	Vascular lesions.
	Acute (few hours or days)	Inflammation.
Pressure and growths	*Subacute* (one to six weeks)	
	Subchronic (six weeks to six months)	Degeneration.
	Chronic (more than six months)	

A lesion of sudden occurrence, the symptoms developing in the course of a few minutes, is almost always vascular, commonly hæmorrhage, sometimes perhaps vascular obstruction. But a vascular lesion may occupy a somewhat longer time in development—a few hours or days. In acute and subacute inflammation the symptoms come on in the course of a few hours, a few days, or a few weeks. Subacute and chronic inflammation occupies from a few weeks to a few months Degeneration, in which there is no adequate evidence of any inflammatory process, occupies many months, or it may be years. The symptoms produced by tumours which invade or compress, and by simple pressure (traumatic causes excluded) are never sudden or very acute, and rarely very chronic, the time occupied by the development of the symptoms varying, according to the nature of the cause, from a fortnight to six months.

It is necessary to consider, however, not merely the whole time occupied by the development of the disease, but also the uniformity of its course. Two or more morbid processes may concur. An initial myelitis, for instance, may lead to a secondary degeneration; and, on the other hand, in degenerated tissues, sudden vascular lesions occasionally occur. Pressure produces local myelitis, which may be independent of the pressure in its development, and have an acute or subacute onset. The whole course of the disease must be ascertained before an inference is drawn, and the possibility of a double process must always be kept in view.

The onset and course of the symptoms thus sometimes enable us to decide at once that a lesion is of a given character, as that one which occurs instantly is vascular, or that one which takes years for its development is degenerative. More frequently they enable us to exclude certain morbid processes, and to restrict the possible lesion to two or three forms. For instance, a lesion which comes on in the course of a few hours must be either vascular or inflammatory. Between these we have to decide by attention to other indications.

In actual diagnosis it is convenient to consider next the indication afforded by the position and distribution of the disease. We consider what diseases occur in this situation, and then which of them have the mode of onset that has been ascertained. This indication involves a knowledge of the various diseases and their seat. The most important consideration is that a wide range of symptoms of uniform character indicates the affection of a definite system of structure, and in most instances a disease commencing in the nerve-elements, and if the onset be chronic we may feel sure that it is a degeneration. On the other hand, the involvement of many functions suggests a random process, such as inflammation or pressure. But this indication is always to be subordinated to the mode of onset. Thus the limitation to a single structure does not exclude inflammation: this may affect, for instance, the anterior grey matter only, and cause corresponding symptoms.

The symptoms may indicate a morbid process limited to one half of the cord, but this does not materially modify the diagnostic method. Almost any process may, in rare cases, be thus limited. System degenerations and acute inflammations are least frequently unilateral, and they never reach a considerable degree of intensity on one side without some affection of the other side. On the other hand, tumours and foci of chronic myelitis are often one-sided, and still more often affect one half of the cord first and then the other.

Indication of disease outside the cord, irritation of certain nerve-roots, causing severe local pain, often precedes the symptoms of compression, and is an important aid to diagnosis. It shows the existence of a morbid process outside the cord before the cord is involved. But we cannot use even this indication except in dependence on the mode of onset. A disease, as a growth outside the cord, may, as we have seen, not only compress the cord, and cause slow loss of power; it may excite inflammation and cause rapid palsy.

The last element in the pathological diagnosis is the detection of any influence which can be regarded as the cause of the disease in the spinal cord, or any associated condition which may indicate an active morbid process. We have seen that the mode of onset may help us to limit the disease to certain possible forms of lesion; the distribution of the affection may render it probable that it is one or other of these forms; and the detection of a cause and the knowledge of the lesion that cause produces may help us to carry the diagnosis still further. The most important general causes of disease of the cord, and the processes to which they chiefly give rise, have been already mentioned. The causal element in diagnosis is chiefly an application of those facts.

The morbid process outside the spinal cord that most closely simulates its disease is, unquestionably, parenchymatous multiple neuritis. The diagnosis depends on a thorough knowledge of the varied symptoms of the latter, since its elements vary according to the different manifestations of the disease of the nerves. For these, and for any general diagnostic principles, the reader is referred to the account of that disease.

The only sure ground for diagnosis is a thorough knowledge of the various morbid processes and their symptoms; and the only safe plan is to work by these, from symptom to seat and onset to nature, treating every case as a problem to be worked out to a definite diagnosis, and only then comparing the result with the types of disease. To one of these the case may or may not conform; if it does not, the comparison with types as a means of diagnosis will only leave the observer stranded and powerless.

The distinction of functional and nutritional disease from organic lesions may conveniently be postponed until the symptoms of the former are specially described.

SPECIAL DISEASES OF THE SPINAL CORD.

DISEASES OF THE VERTEBRAL COLUMN.

Diseases of the bones of the spine fall for the most part within the province of surgery. But there are few of these diseases that do not, among their most frequent effects, interfere with the functions of the spinal cord. Hence an account of the diseases of the cord would be incomplete without some mention of the morbid states that begin in its bony case.

INJURIES OF THE SPINE.

Injuries to the spinal column may consist of punctured wounds (which need not be considered here), concussion (the effects of which on the cord will be considered later), and fracture or dislocation of the vertebræ. It may, however, be useful briefly to describe the more salient features of the last two conditions, their relation to the damage the cord sustains, and the symptoms thus produced.

Dislocation.

Simple dislocation occurs in the cervical region, most frequently at the fifth and sixth vertebræ. It may take place gradually or suddenly: gradual displacement is always secondary to disease of the bones; sudden displacement may occur in disease or from injury. The damage to the cord is always greatest in traumatic displacement of healthy bones, because the force needed to produce the dislocation is much greater, the displacement is more considerable, and the effect on the canal and the contained cord is greater. Displacement may occur in any direction: it usually involves both vertebral articulations, but, in rare cases, it is oblique, involving one articulation only. The common causes are violent blows or falls on the head, rarely sudden rotation of the head while a weight is carried upon it. The symptoms are a lateral or forward or backward displacement of the head, so that the chin is in contact with the shoulder or the chest, or the occiput with the nape of the neck. There is also irregularity of the vertebral spines, usually readily detected. The cord is damaged in most cases, and the symptoms are those of a total transverse lesion in the situation of the luxation. It may be merely compressed, especially in cases of disease, in which the displacement has occurred with little force. The symptoms of paralysis have been known to pass away, in such a case, on the reduction of the dislocation. More commonly the cord is also bruised, with extravasation of blood, and secondary myelitis occurs later. In such cases, if the patients live, there may be anæsthesia or hyperæsthesia below the lesion, with total paralysis of the limbs, and excess of reflex action. In rare instances the cord has been completely divided. In still rarer instances of slight displacement it has not been injured.

Rupture of the transverse ligament which retains the odontoid process may permit the latter to compress the cord, and thus to cause instant death. This often results from sudden suspension by the head, as in criminal executions. One of the curiosities of surgical literature is a case related by Petit in which a man, playing with a neighbour's child, lifted it up by the head, and caused instant death by rupturing the transverse ligament. The father of the child, entering at the moment, stabbed the man with a knife, the blade of which passed in between the first and second cervical vertebræ, divided the spinal cord, and the man also fell dead. Rupture of the ligament has also resulted from raising a heavy weight with the head. The treatment of dislocation is too purely surgical to be described here.

FRACTURE.

All organic diseases of the bones, weakening them, predispose to fracture. Among these, one is of special medical interest; the vertebræ, especially in the lumbar region, may share the rarefaction and weakening of the osseous tissues occasionally produced in tabes, and then a very slight traumatic influence, a blow or wrench, may cause fracture. The force needed is often so slight as to fail to cause displacement or other symptoms except local pain, and symptoms may only supervene at a somewhat later date. Apart from disease, the accident is most common in adults, the greater elasticity of the vertebral column in children giving to them a comparative immunity. Its cause is a blow or fall on the spine, or sudden forcible flexion. In extremely rare cases a fracture, usually slight, has resulted from a severe muscular exertion. This is an important fact, showing that muscular exertion may injure the spine, and may lead to symptoms at a later date. Symptoms of such injury, in slight degree, are not uncommon.

Fracture may occur at any part of the spine, but is most frequent at the fifth or sixth cervical, and at the last dorsal or first lumbar vertebræ. In the dorsal and lumbar regions, the bodies are broken in two thirds of the cases, but in the cervical region, the arches alone are fractured in one half. Usually there is a displacement of the vertebral column at the seat of fracture. Rarely the bodies may be crushed without displacement. The displacement of the bone involves a narrowing of the canal and, usually, compression of the cord. This may also result when the arches only are driven in. But the cord may be seriously damaged when there is no permanent narrowing of the canal, as in Figs. 85 and 86. The dura mater is rarely torn except by a splinter. Blood is almost always extravasated outside the dura mater, often in considerable quantity, from the rupture of the large veins in this situation. There are usually only small extravasations in the pia mater. The cord is, in most cases, bruised and compressed by the lower fragment (Fig. 87). Sometimes it is flattened, and it may even be divided, all nerve-substance being squeezed out of the pia-matral sheath at the spot. In the case shown in Figs. 85 and 86 the cord appeared to have been split longitudinally at the spot, perhaps by the nerve-force of the concussion. It is very common to have local myelitis, opposite the fracture, without any permanent narrowing of the canal or compression of the cord, the inflammation seems to be the direct result of the concussion. Blood may be extravasated into the bruised part, sometimes in minute spots, sometimes in larger hæmorrhages, and even into the central canal. These changes are usually limited to the spot directly damaged, but secondary myelitis may be set up, and sometimes extends beyond the contused area. It

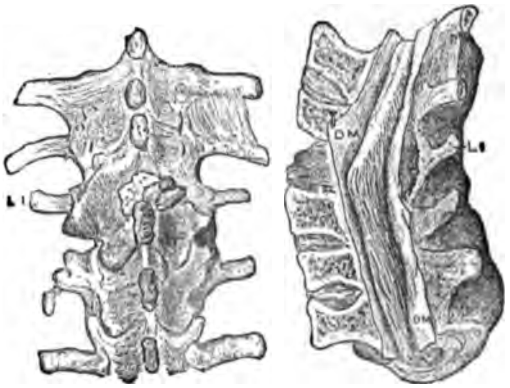

FIG. 85.—Fracture of the first lumbar vertebra. D M. Dura mater. There was no permanent narrowing of the canal, but, nevertheless, the spinal cord was greatly damaged at the spot; see next figure.

FIG. 86.

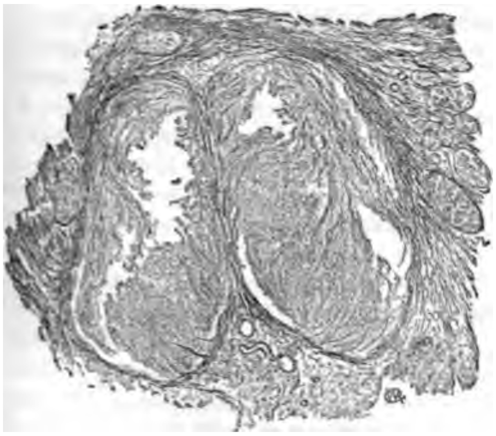

FIG. 87.

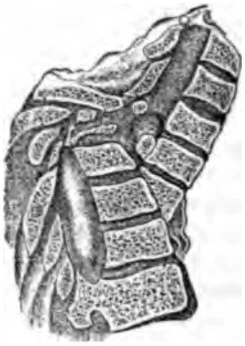

FIG. 86.—Spinal cord damaged by the fracture shown in Fig. 85. The elements of the cord itself are changed beyond the possibility of identification. The ascending degeneration is shown in Fig. 74.

FIG. 87.—Fracture of the body of the fifth dorsal vertebra and of its processes. (After Gurlt.)

may even extend through the whole length of the cord below the injury, and then its central functions are abolished (see p. 228). In cases of some duration the usual ascending and descending secondary degenerations are also found. It is important to note, moreover, that the secondary degenerations have sometimes the irritative character already described (p. 229, *note*), and that an ascending inflammation may for a short distance above the lesion be limited to a tract that degenerates downwards (see p. 232). Fig. 74, p. 177, represents sections of the cord from the case of fracture figured above. Occasionally the injury leads to secondary caries of the bone, with all its consequences.

SYMPTOMS.—Three classes of symptoms result. (1) The local indications of the injury to the spine. (2) There may be certain nervous symptoms not distinctly due to the damage to the cord. One of these is general shock, which may be so great as to entail transient loss of consciousness. Vomiting occasionally occurs. There is great pain in the position of the fracture, rendered very intense by pressure, and often radiating along the nerves which come from this part, the roots of which are compressed. In rare cases epileptiform convulsions have followed fracture, usually at an interval of some days.

(3) Symptoms which result from the damage to the cord, and consist in paralysis of the parts below the injury. Its character depends on the amount of damage. If this is considerable, there is both motor and sensory paralysis up to the level of the lesion, with loss of power over the sphincters. Reflex action is lost at the level of the lesion, and the examination of the trunk-reflexes often gives important

information regarding the extent of the damage, when this is in the dorsal region. Below, reflex action is commonly in excess, unless the centres are impaired by descending myelitis. Spasmodic twitchings sometimes occur in the limbs immediately after the injury, accompanied by priapism. The pains are severe in the arms when the fracture is opposite the cervical enlargement, and in the legs when at or below the lumbar enlargement, so as to damage the nerve-roots. In these cases there may be rapid wasting of the muscles, with loss of electric irritability. There is usually at first incontinence, afterwards retention of urine, but the former is persistent if the lumbar centres are damaged. Cystitis, bedsores, &c., may supervene. Ultimately, if the damage is above the lumbar enlargement and the patient lives, there may be increased myotatic irritability in the limbs, progressing to spasm, so that spastic paraplegia results.

Special symptoms result when the injury is in certain parts of the spine. Fracture of the *first two cervical vertebræ* causes instant death, unless the displacement is very slight, and even then there is imminent danger of further displacement, with the most serious consequences, on any incautious voluntary movement. With slight displacement persons have been known to live for weeks, and then die from secondary myelitis; they have even recovered altogether. Now and then there is no compression of the cord, although there is distinct displacement, even sufficient to be recognised in the pharynx (Leyden). In such a case death has resulted at a later period from caries. The characteristic symptoms are local pain, increased by all movements (which are rendered almost impossible), displacement, and spinal symptoms. The latter may be slight—merely difficulty in breathing or swallowing—or considerable, and involving the trunk and limbs. Sometimes there is hyperpyrexia. Not more than one case in fifty recovers.

Middle Cervical Vertebræ.—The third, fourth, and fifth vertebræ are most frequently fractured. When the injury to the cord is considerable, death usually occurs very rapidly, because the roots of the phrenic nerve are involved, the intercostals being necessarily paralysed with the parts below. In some cases there is little immediate displacement, and the symptoms are slight until further displacement occurs in some movement. Thus a man who had met with an injury of this kind went to be shaved; during the proceeding, his head was turned on one side by the barber, with the unexpected result of causing displacement of the fracture, and immediate death. When the fracture is at the *cervico-dorsal* region, opposite the lower part of the cervical enlargement, the arms frequently escape at first, the early paralysis being confined to the legs and muscles of the trunk. Respiration is diaphragmatic only. After a few days the arms become involved, but their paralysis is often partial, affecting, for instance, only certain muscles, as the extensors of the hand, and it is often accompanied by local spasmodic movements. Movements and pressure cause pain, and there is local muscular rigidity. The head may be in normal or in abnormal position. Vaso-motor disturbance in the face and general hyperpyrexia have been observed.

In fracture of the *dorsal vertebræ* (2—11) the arms escape, the legs are paralysed, and the trunk-muscles up to the height of the lesion. The pain in the trunk may be very severe. There is hyperæsthesia or anæsthesia in the parts below. The reflex action in the legs is excessive; that in the trunk is abolished at the level of the lesion. The *last dorsal* and *first lumbar* vertebræ are fractured more frequently than the others, and there result paralysis of the legs, complete or irregular, severe pains, tingling, &c., sometimes followed by hyperæsthesia or loss of sensibility, and by rapid disturbance of nutrition, in both the muscles and the skin. In fracture of the *lower lumbar vertebræ* the symptoms are often slight; below the extremity of the cord the nerves occupy a smaller space in the canal, and so may escape compression by a moderate displacement. If they suffer, the fractured vertebræ unite very slowly. A false joint is occasionally formed. Secondary myelitis and its consequences are frequent causes of death at a variable period after the injury.

CARIES OF THE SPINE.

Caries of the bones of the spine is a frequent cause of paraplegia. It is often termed "Pott's disease," from the English surgeon, Percival Pott, who first described it (in 1779) as a cause of paralysis.

Causes.—Males are said to be rather more liable than females, but the difference in sexual incidence is not great. It is more common in childhood (after three), and next in early adult life, but it may occur at any age, and is perhaps more common in the second half of life than any other scrofulous lesion. I have known it commence at fifty, and it has been met with as late as seventy. It is distinctly a manifestation of the tubercular and scrofulous diathesis, and evidence of such inheritance is to be traced in most cases. Occasionally the sufferer himself presents such indications, lung disease, &c., or caries of other bones. It occasionally develops simultaneously with other signs of acute general tuberculosis. Injuries seem frequently to excite the bone mischief in the spine, as they certainly do analogous bone disease elsewhere, in those who are predisposed; possibly, sometimes, in healthy persons. Falls, blows on the back, and severe strains are the most frequent traumatic antecedents. The last may act by straining the ligaments and setting up inflammation, which spreads to the bones directly or through the invertebral cartilages. There is usually an interval, sometimes of many months, between the injury and the definite symptoms of bone disease.

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Other causes besides scrofulous disease have occasionally been known to cause breaking down of the bodies of the vertebræ with angular curvature and all its consequences. Soft growths in the spine, and, in rare cases, syphilitic formations have had the same effect. Osteitis, due to extension from adjacent inflammation, is a rare cause; thus caries of the cervical spine has been secondary to a primary retro-pharyngeal abscess. The disease sometimes follows septicæmia, probably in consequence of a secondary septicæmic abscess in the bone. This was the case in a surgeon about sixty years of age, in whom symptoms of blood-poisoning followed an injury to the ankle. They were succeeded by paraplegia of rapid onset, and this by the development of angular curvature in the lower dorsal region. In a large number of cases no exciting cause can be traced.

Pathological Anatomy.—The changes in the bones need not detain us, since they belong to surgical pathology, except in so far as they lead to damage to the cord. There is inflammation of the bodies of the vertebræ, and of the intervertebral substance, often at first with enlargement, afterwards with breaking down of the substance of the bone, which gives way under the pressure to which it is exposed, so that deformity of the spine results. Products of inflammation and destruction of tissue, more or less purulent in character, accumulate either inside the canal or outside the vertebral column. In the latter situation they are increased in quantity by the inflammation they excite and the well-known forms of abscess result.

Within the canal the inflammatory products have a firmer consistence. Although the inflammation extends to the loose cellular and adipose tissue between the bone and the dura mater, and often to the dura mater itself, it has little tendency to spread as a purulent meningitis, or even to penetrate the dura mater, the outer layer of which becomes irregularly thickened in the neighbourhood of the disease (see Fig. 89), while the inner surface of the membrane remains normal. The firm consistence of the inflammatory products, often caseous in character, is an important pathological feature, because this material is so often the agent by which the cord is compressed.

The displacement that results from the disease varies according to the extent and character of the disease. Occasionally there is simply a lateral displacement—one spine is a little to one side of that above it. More commonly the collapse of the bodies leads to "angular curvature," the spinal column is bent forwards at an acute angle, and one or two vertebral spines are much more prominent than the others. There is often, however, a less abrupt bend; the curvature may extend over four or five vertebræ. Less commonly one spine projects more than the others within any curvature.

The *nerves*, as they pass through the membranes and intervertebral foramina, are irritated by the inflammation, and often compressed by the thickening of the dura mater which sheaths them by the bone, and those passing by the seat of compression may also suffer from the narrowing of the canal. They may be found red and swollen, or shrunken and grey. They may be damaged when the cord is normal, or but little affected when this is compressed.

The damage to the *spinal cord* depends on the secondary consequences or the caries, and is variable and uncertain in both occurrence and character. The mechanism of the damage is twofold—compression and inflammation. The relation between the two is considered more fully in a subsequent chapter ("Compression of the Spinal Cord"). Slow compression may be attended by chronic inflammation, secondary not only in character but in course, or by an acute inflammation out of proportion to the compression, alike in degree of severity and in rapidity of development. It is important to recognise these differences. The mechanism of compression varies. The most frequent is the collection of inflammatory products outside the dura mater and the thickening of this membrane. Less commonly the cord is compressed by the displacement of the bone, or by fragments of bone that are pushed into the canal. Often both these causes are influential, as in the case shown in Fig. 88, in which the compression (at S) is between displaced bone in front and inflammatory products (E) behind. Probably an inflammatory swelling or abscess of the bone is sometimes the mechanism of compression, since the signs of pressure have disappeared when an abscess has formed outside the spine, or even when deformity has come on—the breaking down of the bodies having relieved the compression produced by their enlargement.

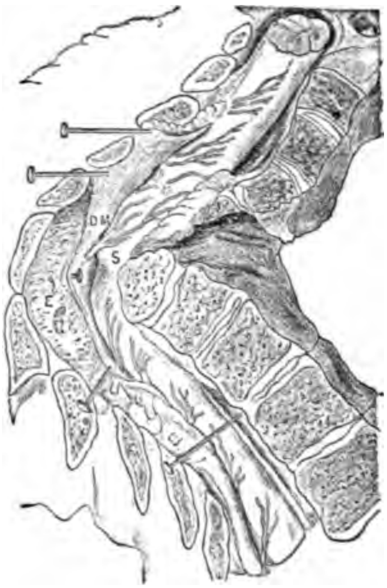

FIG. 88.—Caries of the spine, mid-dorsal region. The spinal cord is much narrowed and discoloured at S from compression between the displaced bone and a mass of inflammatory products, E, outside the dura mater, D M, the inner surface of which is normal. (After Leyden.)

SYMPTOMS.—Caries of the spine causes symptoms of three classes: (1) those of the bone disease; (2) the effects of damage to the nerve-roots; (3) those due to the changes in the cord itself. Only the symptoms due to the damage to nerves and cord come within the special province of this book, but the bone symptoms are of much importance, as on them the diagnosis of the cause of the nervous symptoms often depends. It is this which gives the subject its high medical importance, and brings the symptoms and recognition of bone disease into the province of practical medicine.

Symptoms of Bone Disease.—The first is pain in the spine, chiefly felt at the affected spot, increased by movement, and especially by pressure on the bone. The local tenderness is a very important sign. It is elicited both by direct pressure on the spine and by lateral pressure; if they are grasped and pressed to one side considerable pain is usually produced. Nevertheless tenderness is occasionally absent, not only in cases of slight character and doubtful nature, but also in which paraplegia has come on some time after local curvature. The increase of pain by movement is greatest when the disease is in the more mobile parts of the vertebral column, especially when it is in the cervical region. Movement of the head occasions pain, and there is an instinctive fixation of the head, which is sometimes inclined to right or left, less commonly backwards. It may thus produce the aspect of torticollis, which differs from that due to muscular contraction, in that the sterno-mastoid is tense on the side towards which the head is turned, the muscle being simply stretched by the deviation of the head.

The deformity of the spine is a later symptom than the tenderness, and usually comes on gradually. Its characters have been already described. It is often absent when the disease is in the cervical region. In this part there is usually another symptom—thickening of the tissues about the spine, which is rare in other parts. In any region an abscess may form in the neighbourhood of the disease. Those which come backwards, or descend to the groin by the psoas muscle, can be recognised externally. Those that form in front of the diseased vertebræ may give rise to symptoms that are puzzling, if the existence of spinal caries is not known. Thus a retro-pharyngeal abscess may cause difficulty of deglutition, and one in the dorsal region may cause symptoms of œsophageal obstruction.

Increased pain and tenderness in the vertebral column often precede the indications of damage to its contents. The symptoms due to interference with the *nerve-roots* are very variable, and may be severe or absent; usually they are moderate in degree, and consist of motor pain along the course and in the distribution of the nerves that emerge at the affected region, often hyperæsthesia of corresponding extent, sometimes with spots of anæsthesia. There is also muscular weakness and sometimes muscular wasting. These symptoms are more fully described in the chapter on "Slow Compression." Here it may suffice to say that they are conspicuous only in the minority of the cases, and chiefly occur when there is pachymeningitis. The motor symptoms are most marked when the disease is in the cervical region, and the nerve-roots for the muscles of the arms are damaged, since slight and limited impairment is more readily recognised in the arms than in the trunk. If the disease is in the highest part of this region, the pains may be referred to the occiput. Herpes zoster has been occasionally met with along the course of the irritated nerves. Spasmodic contraction in the muscles supplied by the affected roots is extremely rare in cases of caries. Reflex action is abolished in the affected parts, and the change in the superficial reflexes of the trunk sometimes gives important diagnostic information. Disease of the lower cervical roots may cause symptoms of deranged action of the sympathetic on that side of the head, occasionally shown in the pupil, more often in the vessels. I have seen persistent sweating on one half of the forehead from this cause.

The symptoms of interference with the functions of the spinal cord itself are chiefly due to impairment of its conducting power, causing paralysis below the lesion. As the disease is most frequently in some part of the dorsal region, paralysis of the legs is the common effect. But the symptoms vary in their characters, according not only to the position of the disease, but also to the immediate process of damage to the cord. The signs of caries may have existed for years before paralysis comes on. Angular curvature may even come on in early childhood, and paralysis not till adult life. More commonly the interval between the two is not long, varying from a few months to one or two years, but either may first be conspicuous. The cases are numerous in which caries is not suspected until the paralysis leads to an examination of the spine, and it is not uncommon for the signs of bone disease to remain equivocal for some time after paraplegia has developed.

The cord symptoms vary much in their mode of onset. Usually there is no exciting cause, but sometimes a strain of the back or exposure to cold seems to excite a change in the condition of the bones, in consequence of which the cord suffers. It is easy to conceive that, when the conditions are favorable, a very slight strain may be effective, even as slight as the sneezing in the case mentioned below. When the cord symptoms have commenced, they may develop quickly or slowly. As instances of chronic onset may be mentioned cases in which the symptoms reached a considerable degree of intensity at the end of nine months, four months, and two months, after their commencement. But the onset is sometimes much more rapid; in one case there was complete paraplegia at the end of three weeks. Sometimes it is still more acute, and in such cases, when there is no corresponding change in the bone, acute myelitis set up by slight pressure is probably the mechanism by which the cord suffers. Thus, in one patient, a child of three, slight weakness existed for three weeks, and then the power of standing was lost in a single night. In another child, aged eight, who had presented for two years indications of disease of the cervical vertebræ, the power of moving the legs was lost in the course of twenty-four hours; during the second day the left arm became paralysed, and, at the end of a week, the right arm. Very rarely the onset is instantaneous, probably from sudden displacement. A child with angular curvature was walking across the room, when she fell, and on being lifted up the legs were powerless. The more rapidly compression occurs, the slighter is the amount necessary to abolish conduction.

Both legs usually suffer together; rarely one is paralysed before the other; very rarely one leg suffers much and the other little or not at all. Thus in one case angular curvature developed in childhood; at sixteen there was an attack of weakness in the legs, which passed away at the end of three weeks. At 17½ the patient sprained his back; pain in it followed, and six weeks later the right leg gradually became weak, and a year afterwards presented intense spastic paralysis, the left leg being very little affected. He ultimately recovered. An instance in which one leg became affected before the other is that of a woman, forty-five years of age, who suffered from pain in the spine, and one day, when walking, sneezed violently three times, and immediately felt "pins and needles" about the right knee, and presently in the foot. The leg became almost powerless during the next three days. A fortnight afterwards she felt similar tingling in the left knee, and at the end of another week in the foot, and during the next three weeks this also lost power, so that at the end of six weeks from the onset both legs were motionless. The diagnosis was verified on her death, six months later.

The characters of the paralysis in relation to the seat of the disease are described in the chapter on "Compression." In the most frequent cases the dorsal cord is damaged, and whenever the compression is above the lumbar enlargement the condition of the legs is that of "spastic paraplegia." If it is situated in the cervical region there may be muscular atrophy in the arms, sometimes combined with palsy without atrophy, according to the seat of the disease. When, as is commonly the case, the arms suffer from damage to the nerve-roots, they suffer before the legs, but if the disease is so high up that the arms suffer from the damage to the cord, the legs may be paralysed before the arms, as in the case of the child mentioned above. In this case the muscles of the shoulder were wasted, the disease being at the level of their nerves, while the forearm muscles were not wasted. In this case, as in others of similar seat, the diaphragm was paralysed. The muscular part of the spinal accessory may be involved, and the power of supporting the head may be almost lost. Very rarely, from the disease of the highest cervical vertebræ, other nerves of the medulla are implicated, especially the hypoglossal and the fibres of the spinal accessory for the palate.

Sensory symptoms due to the disease of the cord are less common than motor palsy. The onset of paraplegia may be preceded by subjective sensations in the legs. Dull aching pain in them is also not uncommon. Often there is no loss of sensation; in other cases there are various degrees of loss. Touch or pain may be impaired alone, or there may be absolute loss of sensibility up to the level of the lesion. Partial loss is more common. Reflex action in all forms is excessive (unless the disease involves the lumbar enlargement), and great excess of the superficial reflex action is a common and important feature. The legs are often cold, and sometimes perspire continually. The sphincters are often affected, sometimes early, but they may escape even where there is complete motor palsy of the legs.

Complications.—Among common complications are bedsores, cystitis, various secondary effects of the bone disease, such as abscess, local scrofulous disease elsewhere, and general tuberculosis. Tubercular tumours in the brain occasionally coincide with the caries. In very severe cases peculiar secondary mischief has occurred in the spinal cord, and has run an independent course, giving rise to very anomalous symptoms.* Thus a descending myelitis may invade the lumbar enlargement in its entirety and abolish its central and reflex functions, causing rapid wasting of the muscles and acute trophic changes in the skin. Inflammation may *ascend* the pyramidal tracts and thus paralyse the arms—an instance of the strange limitation of inflammation to functional tracts when it passes in a direction opposite to secondary degeneration. Ascending degeneration of the posterior median columns may spread to the postero-external columns, and cause symptoms of ataxy by invading root-fibres at a higher level. Ataxy may come on as the power returns, when the disease is in the dorsal region, probably by damage to the path from the muscles to the cerebellum. Lastly, myelitis may occur in disseminated foci in various parts of the cord, and even in the medulla, giving rise to scattered symptoms of anomalous character.

Course.—The bone disease may heal, union occurring between the altered tissue, or it may persist with continued formation of pus, or may become quiescent with occasional periods of renewed activity. The cord mischief is influenced by the state of the bone disease, although its progress may be to some extent independent. Thus inflammation in the cord, in excess of the compression, may subside in spite of the continuance of the bone mischief. Pressure on the cord may be relieved, although the bone disease continues, and even sometimes as a result of the increased breaking down of bone and exit of pus by another channel. A case is mentioned below in which paraplegia passed away as angular curvature developed. Hence, there is no strict correspondence between the course of the bone mischief and that of the cord disease. In some cases the paralysis, motor and sensory, persists. More often the sensory loss passes away, while motor paralysis remains, usually as spastic paraplegia, and, in severe cases, flexor spasm comes on with muscular contractions. Life may be prolonged in that condition for years, but often, in such cases, bedsores form, or cystitis leads to kidney disease, or other tubercular disease develops and leads to death, or the lessened respiratory power renders an attack of bronchitis fatal. In many cases, again, the paralysis gradually passes away even when the compression continues, and the cord is found considerably narrowed if the patient dies from some other cause. It is possible that such compression is sometimes produced slowly without impairment of conduction. In children recovery occurs far more readily than in adults. Even in adults, however, recovery may occur from palsy that has lasted for more than a year, with all the signs of descending degeneration in the cord, amounting to severe spastic paraplegia. Usually rest on the back or mechanical supports are necessary to secure recovery, but it occasionally occurs without these measures. A youth acquired angular curvature at sixteen; at twenty paraplegia came on slowly, and progressed, with some variations, during the next seven years. There was then absolute motor palsy in the legs, and sensation was lessened. He refused to rest, and continued to follow his occupation, which was however a restful one, that of a tailor; he took cod-liver oil and iron, and gradually regained useful power, so as to be able to walk about. Such a case, however, is exceptional.

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* See Charcot, 'Leçons sur les Mal. du Syst. Nerv.,' tom. ii.

Relapses sometimes occur in cases that improve, although they are certainly not nearly so frequent as might be expected from the nature of the disease. In the majority of cases recovery, once obtained, is permanent. In a minority the paralysis returns when some exciting cause renews activity in the bone disease. The tendency to relapse and possibility of repeated recovery are very strikingly shown by the following case. In a girl of fifteen, paraplegia developed during nine months, slowly at first, more rapidly towards the end of that time. She came under my care six months later, having been unable even to move her legs for that time. Bone disease had not been previously suspected, but there was slight tenderness and enough lateral irregularity of the lower dorsal spines to show the nature of the case. Rest in bed and tonics were soon followed by improvement; in six weeks she could stand, and in four months was able to walk well. As she gained power, angular curvature came on, prominence of the seventh and eighth dorsal spines. Five months after her discharge, she fell and struck her back; the curvature increased, and her legs gradually became weak again. Seven months after the fall she was readmitted, unable to stand, although the paralysis was not absolute. There was foot-clonus on each side. Sensibility was lessened below the ensiform cartilage. Rest on the back was again followed by slow improvement. In three months she could just walk. She was then suspended, and encased in plaster of Paris. At the end of another month she could walk about the room, and no clonus could be obtained. She was soon afterwards discharged, and her progress continued, so that, at the end of nine months, she could walk five miles, and there was no trace of clonus, although there was still some excess of the knee-jerk. She soon afterwards married, and bore a child, which died two years and a half after her discharge. She caught cold at the funeral, and a fortnight later again began to lose power; in six weeks the legs were almost motionless, with marked foot-clonus; sensation to touch was lost up to the umbilicus, that of pain being preserved. Neither rest nor encasement caused any improvement. After some months sulphide of calcium was given, and in a few days power began to return; in a month she could take a few steps, and in four months she could walk about the ward without difficulty. She made another good recovery. Some years later, however, paralysis again came on, and this attack proved permanent.

Several cases have come under my notice in which the subjects of caries in early life, which healed without damaging the cord, have at some period in adult life presented the symptoms of primary lateral sclerosis—simple spastic paraplegia, without any root symptoms or evidence of renewed activity of the bone disease. If there is a connection between the two, it is probable that the cord has suffered compression so slowly that its functions have not been interfered with, but, nevertheless, the vitality of the pyramidal fibres has been rendered less enduring

It is important to remember that the deviation is often lateral,* and the chief difficulty is due to the fact that a slight lateral deviation or slight prominence is not unusual in normal spines. Hence it is important for the student to make himself familiar with the degrees of deviation that occur in health. At the same time it must not be forgotten that a deviation not greater than occurs in health may be due to disease. If it coincides with distinct tenderness, and especially also with the position of root symptoms, it may be accepted as evidence of disease, probable or certain, according to the character of the symptoms.

The pathology of the affection only concerns us so far as it relates to the effect on the spinal cord, and this is considered in the chapter on Compression.

Diagnosis.—When clear indications of caries precede the paralysis, the nature of the case can hardly be mistaken. The obvious inference, that the affection of the cord is secondary to that of the bone, is scarcely ever wrong. When the two develop together, mistakes in diagnosis are often made, but are usually due to the want of *repeated* examinations of the spinal column It is when the root or cord symptoms precede distinct evidence of bone disease, and when the latter is so slight as to be equivocal, that the chief real difficulty in diagnosis occurs; the affection is apt to be mistaken for a primary disease of the cord or its membranes—a transverse myelitis when the dorsal region is affected, a progressive muscular atrophy, or primary pachymeningitis, when the disease is in the cervical region. A correct diagnosis can only be made in these cases by recognising the significance of the slight bone symptoms that are always present, the deep tenderness, and often slight irregularity. Even slight irregularity derives

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* To discover lateral deviation it is well to make an ink-dot on the skin over the middle of the tip of each spine, care being taken that the skin is not stretched to one side. The ink-dots may be copied on tracing-paper.

[illegible] in extent and corresponding in [illegible] The irregularity may be absent at first, [illegible] significant. An observed increase [illegible] gives significance to even a slight [illegible]

[illegible] reflex action (from the sole, for [illegible] adds weight to the other symptoms of [illegible] even while the patient is able [illegible] are always of great significance, [illegible] or spots of anæsthesia along [illegible] additional weight from their [illegible] irregularity.

[illegible] distinguished from the condi- [illegible] frequently mistaken—a trunk neuralgia. [illegible] common when the pain is chiefly unilateral. [illegible] carefully examined, and any [illegible] the pain should excite suspicion. [illegible] abdominal pain due to caries to be [illegible]

[illegible] causes muscular wasting in the [illegible] mistaken for one of progressive muscular atrophy, [illegible] distribution of the wasting, in the pains, and the [illegible] sensation. These occur in primary cervical pachy- [illegible] distinguished by the absence of the signs of bone [illegible] wider extent through which the root symptoms [illegible]

[illegible] the recognition of bone disease is practically [illegible] recognition of caries. In the second half, however, [illegible] frequency of caries, the greater frequency of growths in [illegible] occurrence of eroding aneurisms, introduce a fresh [illegible] The absence of any other indication of a tumour [illegible] is the first distinction; and the second is the fact that [illegible] diseases the root pains commonly reach a degree of [illegible] attained in caries, and are especially increased [illegible]

[illegible] are merely tenderness of the spine and slight weakness [illegible] question may arise whether there is organic disease, or [illegible] termed "spinal irritation," or mere functional [illegible] tenderness and weakness of the legs. In these cases the [illegible] over a considerable area of the spinal [illegible] one point of special intensity; it is superficial [illegible] may change its seat; there are no root pains or [illegible] There is more danger that caries of the spine in [illegible] may be passed as hysterical paraplegia than of the [illegible] Especially when the subjects of caries present [illegible] of hysteria, there is risk, as experience shows, that [illegible] symptoms of caries may be overlooked. Some other diagnostic indications are mentioned in the chapter on Slow Compression.

Prognosis.—Our ignorance of the precise character of the morbid process which is damaging the cord renders the prognosis, in every case of caries, a matter of much uncertainty. Nevertheless there is no disease of the cord in which symptoms of equal gravity so often pass away. The cases are few, therefore, in which hope is unjustified, but they are equally few in which we are warranted in a confident expectation of recovery. In childhood the prospects of recovery are certainly better than in adult life, and they are least in declining years. Damage to the cord between the enlargements is less serious than when these are affected, because the strong tendency to trophic changes constitutes a grave danger when the lumbar enlargement is diseased, and the diminished breathing power an equally serious danger in disease of the cervical enlargement, especially when this is high enough to entail the additional danger of paralysis of the diaphragm.

But how perilous a condition may be recovered from is shown by the fact that the child mentioned on p. 247 had paralysis of all four limbs, the diaphragm, and weakening of the intercostals, and yet recovered. Still more striking is a case narrated by Dr. Buzzard, in which disease in the region of the third cervical vertebra caused almost complete palsy of arms, legs, intercostals, and diaphragm, respiration being carried on by the accessory muscles of the neck. Yet the child recovered in spite of the occurrence of an attack of pneumonia when the paralysis was at its height. A girl of thirteen, whose cervical caries was accompanied by all the symptoms of cerebellar tubercle, recovered. All these, it will be noted, were children. Neither rapidity nor slowness of onset affords any guide to prognosis, nor does the relative order of paralysis and curvature, or the degree of palsy. Severe spastic paraplegia may pass away entirely, provided it remains extensor in character. The prognosis is perhaps a little better when there is no loss of sensation, since this proves that the damage to the cord is moderate in degree; but even complete anæsthesia does not preclude recovery, as the cases mentioned show. The danger to life is dependent in considerable degree on the evidence that the scrofulous or tubercular tendency is active elsewhere, and also on any difficulty in securing proper treatment. The prognosis is, moreover, at present in a transitional state on account of the uncertainty regarding the range of successful surgical treatment.

Treatment.—The first and chief element in the treatment of paralysis is that of the bone disease which causes it, and for full details of this the reader is referred to treatises on Surgery. It includes both the older means of securing an arrest of the morbid process, and, secondly, the still recent measures of operative treatment. If the bone disease heals, the spinal cord, in most cases, will recover. The two most potent therapeutic agents are persistent recumbency and tonics, especially cod-liver oil and iron. In the cases that have done best, these, and these alone, have been the effective agents. Rest should be maintained for months. The posture that answers best is upon the back, on account of the greater ease with which immobility of the bone is secured and maintained, and the great importance of this element in treatment; this position is also the most comfortable, and local pain probably interferes with the subsidence of local inflammation. If no improvement occurs after some months' rest, suspension may be tried. It is said to be sometimes attended by instant improvement. especially in children; but this result is rare, and can only occur in the cases, not very common, in which displaced bone compresses the cord. In cases in which there is reason to suspect that this is the cause of paralysis, it may be well to see the effect of suspension before rest is commenced. The moulded jacket is an inefficient substitute for rest, and an unnecessary concomitant—to be adopted only when rest cannot be secured, or after rest alone has apparently done all that it can achieve. If, for instance, power gradually returns during rest, but the improvement after a time ceases, and the bone disease seems inactive, the patient may be suspended, encased, and then permitted to stand and walk; improvement may be renewed and power may rapidly increase. In caries of the mobile cervical spine, even during the period of rest, fixation of the head is necessary. Extension of the spine in the recumbent posture has also been employed, sometimes with apparent benefit. The head of the patient is fastened to the head of the bed by an elastic band attached to a strap passing beneath the occipital protuberance and round the patient's head. A weight of from 4 to 12, or even 20 lbs. is fastened to a band passing round the patient above the hips. Continuous extension is thus obtained.*

The influence of cod-liver oil and iron is as marked in this as in other scrofulous diseases. They constitute an indispensable adjunct to rest, and may be effective even alone, as is shown by the case mentioned on p. 250. But rest should always be secured if possible.

Counter-irritation, opposite the seat of caries, has sometimes appeared to do good, perhaps acting especially on the process of myelitis which attends compression. The actual cautery, in mild form, is that which has been most frequently found useful. Instances of speedy improvement are on record, but it is not always effective, nor can indications for its use be laid down. Sulphide of calcium, which is said to influence scrofulous processes, deserves further trial, although I have not found it so apparently effective in any other case than that mentioned on p. 251. Benefit can only be expected from its use when, as is often the case, inflammatory products constitute the compressing agent. Extreme care in general management, to avoid bedsores, bladder trouble, and bronchitis, are of great importance. In many cases there is a tendency to improvement after a time, especially when the bone disease is stationary, or the cord has been inflamed, and in these the mere maintenance of life may result in recovery of strength. When power has returned, but the use of the limbs is restrained by the muscular contractions which came on during the paralysis, the ability to stand and walk may be quickly restored by tenotomy, and more slowly by the use of extending splints, &c. Electricity is useful only to maintain the nutrition of the muscles when these are wasting, in consequence of damage to their nerves.

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* Fleming, 'Lancet,' April 27th, 1889.

The belief expressed in the first edition of this work, that the cases are numerous in which relief to the compression may be afforded by the surgeon, has been fully realised. The path was, indeed, being then opened up by McEwen of Glasgow, who has shown, with Horsley and others, how safe and successful the operative treatment is in properly selected cases; and these, indeed, include a large proportion of the cases in which other measures have failed to give relief and must probably have remained unsuccessful. The operation is contra-indicated in cases of rapidly advancing general tuberculosis (to which most deaths after operation have been due). When the indirect consequences of the disease have greatly reduced the patient's strength, the question of an operation may still be entertained if it is a distinct alternative to a speedy death, provided the peril is such as a restoration of the functions of the cord might possibly relieve. On the other hand, the operation ought not to be regarded as the first resort in the majority of cases. The large number of cases that recover under the treatment described above, make it certain that, in most cases, these measures should first be tried; and this conclusion is the clearer, since the duration of paraplegia, for a year or more, seems to interpose no appreciable barrier either to recovery or to the relief that can be given to the pressure. On the contrary, indeed, the prospect of recovery is better if the bone disease has become quiescent, or the caries has actually healed, processes that must be furthered by the preliminary rest. The operation has the best prospect of success in the cases, fortunately very numerous, in which the products of inflammation outside the dura mater compress the cord. These, whether caseous material or a mass of connective tissue into which the inflammatory products have been changed, can be readily removed without opening the dura mater. Even if displaced bone is the source of pressure, the removal of the arches affords relief to it. The chief obstacle to success is the extent to which myelitis has proceeded out of proportion to the compression. This, necessarily, no removal of the pressure can relieve. In proportion as the inflammation has been acute without rapid compression, relief is unlikely. But this is not the only cause of a rapid onset, and a difficult question arises in such cases, whether the mechanism is not such that an immediate operation is justified. It may be due to sudden displacement or to sudden escape of pus into the canal, as well as to myelitis, and in the two former cases it is possible that relief may sometimes be given, and an immediate operation may be wiser than delay. An operation is suggested whenever a sudden increase in the curvature or severe root pains coincide with the onset. Without these symptoms, the rapid onset is probably due to acute myelitis, and then an operation cannot be expected to lessen the interference with the conducting fibres. To justify surgical interference, however, the existence of caries must be beyond doubt. The operation involves some danger to life, although it may not be great, and this fact must always receive due consideration in regard to the certainty of diagnosis as well as the prospect of relief.

TUMOURS AND OTHER DISEASES OF THE SPINE.

GROWTHS IN THE SPINAL COLUMN.

The bones of the spine are sometimes the seat of primary or secondary growths, and less commonly are invaded by tumours springing from the various structures and even organs in front of the spine. Cancer (scirrhous and encephaloid) and sarcoma are the most common forms of growth; myxoma has been also met with. Cancer is sometimes secondary to a primary growth elsewhere, in the breast, stomach, &c. A primary growth usually begins in the bodies of the vertebræ, and spreads from one to another. The bodies may collapse, because the soft tissue which replaces the bone yields under the weight it has to bear, and thus angular curvature may occur, or some other abrupt deviation from the normal line. From the bodies the growth may extend into the lateral processes, enlarging them, and narrowing the intervertebral foramina through which the nerves pass. It may extend into the arches, and even into the spines, and may also grow into the muscles and tissues beside and behind the vertebral column. The nerve-roots suffer (1) by pressure, (2) by simple inflammation, (3) sometimes by cancerous infiltration. They may thus be found reddened, swollen and soft, or grey and atrophied, or enlarged and hardened. The growth, cancer especially, is apt to spread in the adipose tissue between the bone and the dura mater, and may even entirely surround the dura mater and cord. The cord may suffer compression, but this is less frequent than in caries, even when curvature occurs. On the other hand, it is often inflamed, sometimes acutely; considerable inflammation without compression is far more frequent than in caries. The myelitis occurs without any perforation of the dura mater by the growth. The cause of this tendency to acute local inflammation is not known; once excited, it may spread in the cord upwards and downwards. The growth never invades the cord itself.

ETIOLOGY.—The general causes are the same as those of similar growths elsewhere. Males suffer, however, more frequently than females, and the morbid growths, taken together, are most common between forty and fifty years of age. An injury has been supposed sometimes to be the exciting cause in this as in other situations.

SYMPTOMS.—There may be direct symptoms of the presence of the growth—pain in the spine, sometimes severe, sometimes absent; local tenderness, and occasional interference with movement of the vertebral column apart from pain. A palpable tumour is never an early symptom, but ultimately a deep-seated hard swelling may be felt on one side of the spine, most readily and earliest when the disease is in the cervical region, where it is occasionally perceptible in the posterior triangle of the neck. The angular curvature which sometimes occurs does not differ from that of caries in its external characters, except that it is more frequently a rounded prominence than a sharp angle, and it may be accompanied by the indications of a tumour, never by those of an abscess.

The affection of the nerves is almost invariable in spinal growths, and gives rise to the symptoms that are the most distressing, and also the earliest and most constant,—radiating pain in the course of the nerves that emerge at the part, and due to their irritation by pressure or inflammation. Such pains are far more prominent symptoms in this disease than in caries. At first slight, they gradually increase to extreme intensity. It was this feature that led Cruveilhier to call the disease *paraplegia dolorosa*, a name it has since commonly borne. The pain at first intermits, but subsequently is constant, with paroxysms of greater suffering from time to time. It is usually a sharp and lancinating pain, and its special characteristic is the degree in which it is increased by movement. Even slight movements of the trunk induce the paroxysms. The distribution in the arms, trunk, legs, depends on the seat of the disease. It is extremely rare for these pains to be absent, but they occasionally occur late instead of early.* On the other hand, they may precede other symptoms for months. Cutaneous hyperæsthesia usually accompanies them, and spots of anæsthesia often develop, after a time, in the areas to which the fixed pains are referred. Corresponding damage to the motor roots may cause painful muscular contracture (Figs. 56 and 57, p. 91), paralysis, and wasting. Contractures may also occur in the parts below the disease, produced by the persistent pressure on the motor tract in the cord. Paroxysms of spasm often attend the pains, especially in the abdominal muscles when the disease is in the dorsal region, and are apparently reflex in nature.

The damage to the cord causes symptoms similar to those in caries, and described more fully in the chapter on "Compression." The chief difference from caries is the greater frequency of a rapid onset of the paraplegic symptoms, due to the invasion of the cord by acute inflammation. All power in the legs is often lost in twelve or twenty-four hours, and this when no curvature has taken place. Such acute paraplegia is not uncommonly preceded for a week or so by retention of urine, probably indicative of slight pressure in the cord. Displacement of bone has also been known to cause rapid paralysis.* On the other hand, especially in very slowly growing tumours, the onset of the palsy may be gradual. I have known it to occupy several years in reaching a considerable degree. Probably in these cases the mechanism is a simple slow compression. Thus, compared with caries, the onset is more often rapid, and occasionally much more deliberate. The characters of the resulting paralysis are, as a rule, similar to those in caries, but are more frequently modified by the spread of inflammation, immediate or subsequent. Hence the central and reflex functions of the lumbar enlargement, although the bone disease is some distance above it, may be lost at the onset, or they may be at first normal or excessive, and afterwards lost. In a man with a growth in the mid-dorsal region, paraplegia came on rapidly, evidently from myelitis, and was followed by foot-clonus, &c. But a few weeks later the clonus suddenly ceased, the muscles became toneless, with loss of faradic irritability, and the skin began to slough. The inflammation had spread down into the lumbar enlargement. In many cases, however, there is no descending inflammation, and the reflex actions are in persistent excess, so that spastic paraplegia results. Sensation is lost rather more frequently than in caries. Other symptoms are the same as in compression from any cause. The course of the disease is from its nature progressive; occasionally, however, some improvement succeeds a rapid development of palsy, and is due to the partial recovery of a cord damaged disproportionately by inflammation. It is seldom, however, that life is prolonged for a sufficient length of time. Much more rarely the pains lessen, although the growth spreads—perhaps from destruction of irritated nerves.

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* The pains succeeded curvature in a case described by L. Humphry ('Lancet,' 1884, i, p. 15).

The *duration* of the disease varies according to the nature of the growth. In cancer it is to be measured by months. In slowly growing tumours the symptoms may last for years. Death may be due to bedsores, &c., to cystitis and kidney disease, to growths elsewhere, or the patient may be simply worn out by the prolonged agony. In one curious case a growth from an intervertebral cartilage caused characteristic symptoms for thirteen years, and then death by meningeal hæmorrhage.†

DIAGNOSIS.—The recognition of the disease is only a matter of certainty when signs of a tumour are present, but the probability almost amounts to certainty when such symptoms as those mentioned follow a primary growth elsewhere. Such pain as that above described, occurring in a person from whom a malignant tumour has been removed, should always be regarded with grave suspicion, and the grounds for this are not lessened by either an interval of several years since the removal of the tumour, or by the completeness with which this was effected. Especially is this true of cases of mammary cancer. The pains should lead to careful and repeated examination of the spine and a search for any abrupt deviation, in any kind, and in any part of the vertebral column; while the cervical spine should be examined at the sides and from the posterior triangle of the neck, the character of the bony prominences on the two sides being minutely compared. Indications of a growth may often be thus discovered long before they are obtrusive. It is also important to remember the fact that acute transverse myelitis may be an early effect of the disease, and if this lesion, in such a subject, coincides with marked local tenderness, the probability of secondary growth in the bone is very great; it is rendered still greater by preceding pains, and certain by coincident deformity.

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* Humphry, loc. cit.

† Boivert, 'Arch. de Phys.,' 1887, No. 8.

It must be remembered that similar root symptoms are sometimes due to an aneurism or to a growth in front of the vertebral column, commencing, for instance, in the glands, irritating the nerves as they emerge from the intervertebral foramina.

From intercostal neuralgia, the influence of movement on the pain is usually a sufficient distinction, even when cord symptoms are absent. The commonly bilateral character of the pain is a further difference. The symptoms of a growth in the lower cervical region are sometimes very closely simulated by symptoms which, from their character and course, are probably due to radicular neuritis. There are severe root-pains, referred to one arm (or less commonly, to both arms), increased by movement and without marked tenderness of the plexus of nerve-trunks. The subjects are also in the later period of life. The distinction rests on the absence of the signs of a growth, on the slighter effect of motion on the pains, on the absence of any progressive tendency, and on the common presence of a history of gout, and especially on the absence of any symptoms of compression of the cord even after the nerve-roots have suffered for some months. Sometimes it is necessary to wait and watch the course of the symptoms. It is necessary to be very careful in assessing the value of slight irregularities; the vertebra prominens is apt to be thought to project too much even for its designation, and a deep bilateral swelling is readily perceived under normal conditions, when the head is bent forward.

The chief difficulty in diagnosis is the distinction from caries when distinct evidence of a growth is absent. In the first half of life caries would alone be thought of in such a case, but in the second half the two diseases are about equally frequent. One distinction, suggestive, not absolute, is the intensity of the pain in tumour, taken in conjunction with its great increase when the patient moves. It is true that the root-pains of caries are said to be sometimes most severe, but such severity is not frequent enough to destroy the significance of intensity. I have not, for instance, seen a single case of caries (of a large number) in which the pain was comparable to that in most of the cases of growth that have come under my observation. Therefore, while absence of pain is of slighter diagnostic value (in favour of caries), great severity, and agonising increase by movement, are strongly in favour of vertebral growth. In each disease there may be angular curvature, but this, in growths, is usually soon succeeded by other signs of tumour. In caries these signs are absent, and an abscess often develops. A history or indication of tubercle is almost conclusive evidence of caries. These points will, I believe, avail for the distinction in most cases. In a few it is necessary to wait and watch before an opinion can be formed.

Among other diseases with which these growths may be confounded is the dorsal form of tabes, in which severe radiating pains occur in the trunk and not in the legs. But the wide extent of the root symptoms, the slight effect of movement, and the fact that the knee-jerk is lost, suffice for the distinction. The distinction from tumours of the spinal cord and membranes is considered in a later chapter.

Prognosis.—The prognosis scarcely requires formulating. The chief differences are in the time that life is likely to last. The pains usually persist, in spite of the progress of the disease, although there is a bare possibility of their subsidence. The chance of any return of power in the paralysed part is small, although not quite absent if the palsy develops in a manner to suggest a secondary myelitis, and the progress of the growth itself is slow.

Treatment.—Possibly in a few cases a growth may be so placed as to be removed, and an exploratory operation would be justifiable in any case that might prove suitable; otherwise treatment must necessarily be confined to the relief of pain, and to the avoidance of bed-sores and other results of the cord disease. Morphia is alone powerful for the relief of pain, but unhappily the dose has to be quickly increased, and the power of the drug is lessened by custom. It becomes a race between dose and pain, in which, if life lasts long, the pain not uncommonly gets in front of the narcotic. Cocain, however, affords some relief in many cases, and may, with other anodynes, at least prolong the influence of moderate doses of morphia.

Vertebral Exostoses.

Exostoses sometimes grow from the bodies of the vertebræ into the spinal canal, and may compress the cord or nerves. They are, however, exceedingly rare. The symptoms may be those of slow compression of the cord, or of irritation, expressed chiefly by pain. They usually resemble those of a tumour of the cord or membranes rather than of the bones, but the pain is occasionally much increased by movement. Their chief characteristic is extreme chronicity. In one case the patient suffered frequent intense paroxysms of pain in the right groin, which had occurred for two years, with occasional intervals of freedom. There was some weakness of the legs, but no considerable paralysis. Ten years before, he had had some loss of sensibility in each thigh, which had passed away. An intra-spinal tumour was diagnosed; the post-mortem examination revealed exostoses from the bodies of the ninth and tenth dorsal vertebræ, slightly compressing the cord. Although extreme chronicity may raise a suspicion of exostosis, it is doubtful whether a confident diagnosis is ever justified except in the cases in which there are similar exostoses elsewhere. This rare indication existed in a patient, under the care of my colleague Dr. Barlow, who presented multiple exostoses, and paraplegia of gradual onset, which was supposed during life, and found after death, to be due to a similar exostosis within the spinal canal. It had sprung from one of the lumbar vertebræ, and had compressed the nerves of the cauda equina.

Exostoses constitute a more promising field for the surgeon than other kinds of vertebral tumour. Many of them are so placed that their removal is feasible. If situated in front of the cord, the division of some nerve-roots, at least in the dorsal region, might permit access to the growth.

Syphilitic Disease.

Syphilitic caries of the bodies of the vertebræ is a rare variety, the symptoms of which do not differ from those of the scrofulous form. It has been observed in the cervical region, secondary to deep syphilitic ulceration of the pharynx. Nodes of the vertebræ, within the canal, are occasionally presumed to exist and to compress the cord, but I do not know of any pathological observation confirming the assumption, and it is probable that most of the supposed instances have been cases of syphilitic gummata in the meninges. Deep-seated thickening of the tissues about the cervical vertebræ sometimes occurs in syphilitic subjects. It may develop on one side or both, and is apparently due to a syphilitic cellulitis. The swelling may be felt either on each side and behind the upper cervical spine, or deep in the posterior triangle of the neck. It may damage the nerves before they enter the brachial plexus, causing a defined palsy, as in one case of the lower arm muscles. Movements of the neck may be interfered with, and irritation of the nerves may cause neuralgia-like pain, generally felt down the arm, and often very severe. Except by the absence of nodulation, it is scarcely to be distinguished from a deep-seated growth. The spinal cord does not usually suffer. All the symptoms pass away under antisyphilitic treatment. In one case, however, iodide had no influence, although mercury quickly cured.

Erosion by Aneurism.

Bones, like other structures, may atrophy and waste before the pressure of an aneurism, and the bodies of the dorsal, or rarely of the lumbar vertebræ may be thus eroded by aneurisms of the aorta. The pressure and absorption take place from the left side. Two or three vertebræ usually suffer, and the bodies more than the intervertebral cartilages. The periosteum becomes thickened, and may resist the pressure and to some extent protect the cord. Sometimes, however, the cord becomes compressed, or the periosteum may come to form part of the wall of the aneurism, and may give way before the blood-pressure, so that rupture occurs into the spinal canal.

The symptoms vary much. Pain along the nerve-roots is usually severe, but this may attend aneurisms that merely compress the nerves after their emergence, and do not damage the bone. The process of erosion is usually attended by severe pain in the spine. When the cord is reached, compression causes the usual paraplegic symptoms, of slow or rapid onset. Rupture into the canal is attended by sudden complete paraplegia and death, either immediate, or in the course of a few hours, from ascending paralysis, due to the hæmorrhage around the spinal cord.

The diagnosis is scarcely possible unless other indications of aneurism are detected, since the symptoms closely resemble those of a growth in the bone. The nature of the disease may, however, be suspected if such symptoms as have been described are succeeded by sudden paraplegia followed quickly by ascending paralysis.

Hydatid Disease.

Hydatid cysts sometimes develop in the loose adipose tissue between the dura mater and the bone, and it is believed that they sometimes form in the substance of the bones themselves. About a dozen cases have been collected by Leyden.* As the cyst grows, the bone of the arches sometimes becomes atrophied by pressure, and the cyst may develop outside the canal, so that there is a double cyst, outside and inside, connected by a narrower part. Occasionally the cyst develops also in front of the spine, in the thorax or abdomen. The internal cyst necessarily compresses the spinal cord, which often also becomes inflamed. The usual paraplegic symptoms develop, both motion and sensation being usually lost. Radiating pains along the nerve-roots are frequent. The symptoms, in themselves, resemble too closely those caused by other diseases of the spinal column to permit a diagnosis to be made, unless similar disease elsewhere suggests the nature of the spinal lesion, or unless the cyst can be felt in the back, when a puncture may prove its nature. All the cases hitherto recorded have terminated fatally, but it has been conjectured that, if puncture can be effected, a cure may sometimes be possible. In one such case, however, a girl, aged twenty-two, in which a small tumour beside the last dorsal and first lumbar spines was opened (on account of complete palsy of legs, bladder, and rectum), a continuous discharge of echinococcus cysts occurred, more than a hundred escaping in the course of four months, when the patient died in consequence of ascending damage to the cord. The tumour has been noticed for eight years before death, during four of which it produced symptoms of irritation of the nerves, and finally prominence of the vertebral spines developed.*

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* 'Klinik der Rückenm. Krankh.,' Band i.

Numerous small cysticercal vesicles have also been occasionally found within the dura-matral sheath. In one case, several such vesicles were attached to nerve-roots, one to each, in the lumbar region, while a considerable number on the cervical enlargement were beneath the pia arachnoid.† Such cysts may compress the cord, producing paraplegia, and even death. As in other forms of compression, the paraplegia may come on suddenly.‡

DISEASES OF THE ARTICULATIONS.

LATERAL CURVATURE OF THE SPINE seldom affects the functions of the cord. Even when slight compression has occurred, the slowness of its production has apparently prevented interference with function. In very rare cases some weakness of the legs has been present, possibly, but not certainly, the result of the curvature. Occasionally the intervertebral foramina have been narrowed, and the pressure on the nerves has caused radiating neuralgic pains.

VERTEBRAL ARTHRITIS.—In chronic rheumatoid arthritis the intervertebral articulations are sometimes involved. The symptoms are local pain, tenderness, and limitation of movement, which may go on to absolute fixation, especially in the cervical region, if anchylosis occurs. I have known the whole neck to be rigid from this cause, in a case in which also the movement of the lower jaw was much reduced. More frequently, movement is restricted by pain before the mechanical limit is reached. Sometimes an occasional clicking sound occurs. Enlargement of the ends of the bones may occur, and, in thin persons, may even be felt. The pain is increased by movement, and often by changes in the weather, and by fatigue. It is often felt through a considerable extent of the spine, and may extend to the back of the head. The cord is scarcely ever compressed, but the narrowing of the foramina may damage the nerve-roots. The articular processes, it will be remembered, form one side of the intervertebral foramina, which are thus encroached on by any enlargement of the bones. The nerves may thus be compressed and irritated, and radiating pains are produced, sometimes even a descending neuritis. The nerve-trunks in the limbs may then become tender as well as painful; muscular atrophy may even occur, and, when the disease is in the cervical region, there may be symptoms of disturbance of the sympathetic. Spasmodic torticollis is said to have been caused by this disease in the cervical region. The atlo-occipital articulation has been affected alone, or with the adjacent vertebræ, and peculiar interference with the movement of the head has resulted. Sometimes the foramen magnum is narrowed, or the odontoid process projects into the canal. Symptoms of damage to the nerves of the medulla have also been produced (Solbrig). In the dorsal region the symptoms produced are slighter in degree, but similar in general character, although the local symptoms usually preponderate over those of irritation of the nerves.

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* Pedjkow, 'Med. Obos.' (Russ.), xxviii; and 'Cent. f. Nerv.,' xii, 271.

† Hirt, 'Berl. kl. Wochenschr.,' 1887, No. 3. The relation of the symptoms to the parasitic disease is doubtful in a degree that makes it useless to mention them.

‡ As in a case recorded by Wiegand ('Warsaw Med. Ob.;' and 'Cent. f. Nerv.,' 1888, 665).

The degree in which the morbid process causes symptoms varies, however, very much in different cases, and it has occasionally been found out at the post-mortem examination without being suspected during life. It is usually associated with distinct evidence of a rheumatic diathesis, and commonly with obvious arthritis elsewhere. This is the most important diagnostic indication as to the cause of the spinal symptoms, but it must be remembered that the vertebral affection sometimes exists alone, and it may cause symptoms during many years, often when their nature is unsuspected. It occurs in both sexes, and is not rare in young women, in whom the symptoms are often ascribed to hysteria. The treatment is the same as that of the general disease, nerve-symptoms alone needing special treatment, such as is described in the section on diseases of the nerves. The most important point in regard to the malady is its diagnosis, because distant nerve-pain is liable to be regarded as unconnected with local disease, or the disease to be thought far more serious than it really is, and especially to be mistaken for chronic meningitis or malignant disease.

DISEASES OF THE MEMBRANES OF THE SPINAL CORD.

The general arrangement of the membranes of the spinal cord has been already mentioned (p. 162). The pia mater closely invests the cord, while the arachnoid forms a loose sheath around it, and the two membranes are connected by trabeculæ and membranous expansions of fine connective tissue, which occupy the "subarachnoid space." Each surface of the dura mater is covered by a layer of epithelium. The outer sheaths of the nerves are continuous with this membrane; the connective tissue, within the outer sheath, is continuous with both the pia mater and arachnoid. The blood-vessels of the cord ramify, as we have seen, in the pia mater, and the lymphatic canals of the two are continuous. Most of the cerebro-spinal fluid within the vertebral canal is contained in the subarachnoid space, but there is a little fluid between the dura mater and the arachnoid. Both membranes are supplied with nerves, but those of the pia mater are the more abundant.

The morbid processes that involve the membranes are chiefly three, —growths, hæmorrhages, and inflammation. Tumours of the membranes may be conveniently considered in connection with those of the spinal cord. Inflammation and hæmorrhage will be here described.

SPINAL MENINGITIS: INFLAMMATION OF THE MEMBRANES OF THE SPINAL CORD.

Inflammation of the membranes may be acute or chronic, and may begin, and affect chiefly, the dura mater (*pachymeningitis, i. e.* inflammation of the firmer membrane), or the pia mater (*leptomeningitis,* inflammation of the softer membrane). The arachnoid usually suffers with the pia mater, but is sometimes the seat of inflammation that affects the pia mater but little, a form that has been termed *arachnitis.* Acute inflammation, beginning in one membrane, usually spreads to the others. Chronic inflammation may remain limited to one membrane, dura mater or pia mater.

All forms of acute inflammation, wherever they begin, cause similar symptoms. It is only when the inflammation is chronic that the symptoms may differ sufficiently to allow of a precise diagnosis.

Spinal meningitis may be conveniently divided into two forms, which depend on the situation of the inflammation in regard to the dura mater: (1) that which begins outside this membrane, and sometimes arises by extension from some adjacent focus of inflammation, —*external meningitis;* (2) that which begins within the dura-matral sheath, and is often primary,—*internal meningitis.*

EXTERNAL MENINGITIS.

External meningitis is thus an inflammation of the dura mater. It has been termed *external pachymeningitis*, *peripachymeningitis*, and *perimeningitis*. The inflammation involves not only the membrane, but also the connective tissue outside it, in which are the venous plexuses and adipose tissue. This connective tissue may be inflamed before the membrane itself. The morbid process usually remains limited to the outer surface of the dura mater, even when the inflammation is acute and intense, passing through it only in rare cases.

Causes.—External meningitis is generally due to extension from some contiguous disease, especially of the bones of the spine, caries of which is the most frequent cause of the affection. It results also from other forms of bone disease, and a syphilitic inflammation is probably, in rare cases, associated with syphilitic disease of the vertebræ. Such secondary external meningitis is generally limited in extent and chronic in cause. Deep sacral bedsores, however, sometimes cause an acute inflammation, which may spread widely. An ascending neuritis has been supposed in some cases to set up local inflammation. Suppuration adjacent to the vertebral column is sometimes associated with a general acute purulent meningitis, and has generally been regarded as the cause of the latter; but careful observations have rendered it probable that in most cases the external suppuration has been the consequence, and not the cause, of the meningitis. It is certain that acute general external meningitis occurs as a primary disease, and may run an intensely rapid course with profuse suppuration between the membrane and the bone. The subjects have generally been young, ill-nourished adults, and when any exciting cause has been traced, this has usually been exposure to cold. In some cases the pus has worked its way backwards between the vertebral arches (sometimes through the foramina), and has spread among the muscles of the back, sometimes forming also collections at one or more spots, either at the back, or in front of the spine,—under the pleura, for instance, or behind the peritoneum. Such local collections of pus were also formerly regarded as the cause of the meningitis. It is still possible that this mechanism is effective in some instances, as when a retro-pharyngeal abscess is followed by the meningeal affection; but it is certain that in many cases the actual relation is the reverse, and that the external suppuration, in acute cases, is often secondary.* Hence it is probable that this is true of all cases in which the evidence of primary external disease is not conclusive. At the same time further observations are needed to decide the point.

Pathological Anatomy.—The inflammation is sometimes simple, but much more frequently purulent or semi-purulent. If simple, the dura mater may be merely reddened and opaque, with a little lymph on the surface; when purulent, the surface is covered by a layer of pus. In the more common semi-purulent form, such as is common in caries of the spine, a layer of inflammatory products covers the outer surface of the dura mater, soft, semi-caseous or firm caseous material, sometimes with liquid pus here and there in the firmer substance. Often the inflammatory products undergo development into fibroid tissue, a mass of which then lies between the dura mater and the bones. Such a layer, caseous or fibroid, may sometimes be half an inch thick (see Fig. 88, p. 245). It may either surround the dura mater, or be chiefly on one side. Sometimes there is an irregular nodular thickening of the outer layer of the membrane (Fig. 89). In most cases the inner surface is normal, even when the cord itself is inflamed. Occasionally the inner surface of the dura mater is also inflamed, and then the internal membranes are involved and adhesions form between them.

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* Two instructive cases are recorded by Dr. W. H. Spencer, 'Lancet,' 1879, i, and Dr. Maguire, ib., 1888, ii.

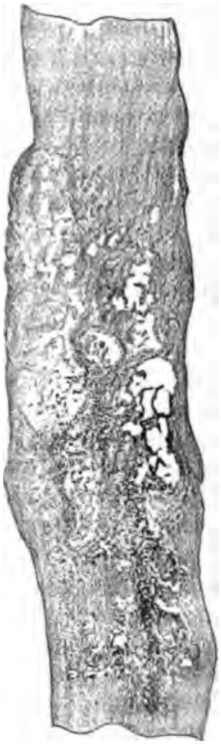
FIG. 89.—External pachymeningitis; nodular thickening of outer layer of dura mater in caries of the spine.

In all forms of external inflammation, the fat outside the dura mater quickly becomes absorbed, and the membrane may ultimately become adherent to the bone, or connected with it by intervening fibrous tissue of inflammatory origin.

The vertical extent of the disease varies much; when secondary to caries, the inflammation is usually limited to the neighbourhood of the bone disease. When acute and general, as in the primary form, the space between the membrane and the bone is filled with pus through the whole length of the canal. Occasionally the pus only lies behind the membrane, between it and the arches of the vertebræ, apparently determined in position by gravitation. It ceases in the upper cervical region, where the dura mater is in closer contact with the bone, its ascent being perhaps also hindered by gravitation as the patient lies with the head raised. As already stated, pus is often found among the muscles of the back (having escaped between the arches), and sometimes collections are found in front of the spine. The amount outside is no doubt increased rapidly by the local suppuration excited by the presence of the pus. In these cases the membrane itself is swollen, and has a soddened aspect, resembling a piece of wash-leather, being softened in proportion to the duration of the affection. The inner surface is usually normal, and the pia mater is either unchanged or merely congested.

Symptoms.—In different cases of external meningitis the symptoms vary much, and they are usually complicated with those of the cause of the inflammation. Those that occur in caries have been already described. In most acute cases the symptoms are nearly the same as those of internal meningitis, although they are subject to greater variations, and more frequently have unusual and anomalous manifestations, presently to be described. The chief symptoms are pain in the back, often referred to the loins, increased by movement, accompanied by stiffness of the muscles of the spine, severe root-pains, and cutaneous hyperæsthesia. The pains are usually associated with spasm; the hyperæsthesia may be followed by anæsthesia, and sometimes by paralysis of the muscles supplied by the irritated and damaged nerves. When there is a rapid formation of pus, the functions of the cord itself are rapidly interfered with in consequence of its compression, and the symptoms of irritation are to a large extent replaced by those of paralysis, beginning in the legs and ascending. The motor palsy is accompanied by flaccidity of the muscles and loss of all reflex actions. Sensation may also be lost, or may be preserved or changed. The sphincters are usually paralysed. Bedsores form rapidly if life is sufficiently prolonged. A peculiar lividity of the skin has been sometimes noted. In these acute cases there is much constitutional disturbance, rigors, profuse sweating, and high fever, in one case amounting to 110° (Maguire).

In local secondary forms the symptoms differ in character according to the acuteness of the inflammation, and in distribution according to its seat. Although, as a rule, pain is considerable, other signs of irritation, stiffness and muscular spasm, are generally inconspicuous, and the symptoms resemble, and are merged in, those due to the disease by which the meningitis is produced.

Diagnosis.—The most important indications of the chronic form are the vertebral pain and the radiating pains, with other symptoms of irritation of the nerve-roots, combined with the signs of pressure on the cord. These symptoms are seldom distinct from those of the cause of the external inflammation; the meningitis is indeed the chief mechanism by which the local disease causes its nerve-manifestations.

The acute form causes symptoms that have much in common with those of internal meningitis. It is indeed doubtful whether the diagnosis can be made from the character of the nerve-symptoms alone. The special indication of the seat of the process is furnished by evidence of its extension to or from the structures outside the spine. If such acute symptoms are secondary to external disease that clearly preceded the meningeal inflammation, this may safely be assumed to be outside the dura mater. In a case of apparently primary meningitis a careful watch should be kept on the tissues of the back; any sign of deep œdema among the muscles beside the vertebral column, in such a case, is probable evidence of commencing purulent inflammation extending from within, and the development of acute local inflammation in either the pleura, posterior mediastinum, back of the abdomen, or behind the pharynx, has the same significance. It is important that the nature of such symptoms should be kept in mind, as otherwise their occurrence increases the perplexing character of the case. It is probable that the nature of many cases, which remain undecided during life, would be rendered clear by repeated careful observation.

PROGNOSIS.—The acute affection is exceedingly grave. Recorded cases have ended in death, but this termination has been a condition of the diagnosis; and it is possible that some cases which have recovered, in which the exact seat of the inflammation was uncertain, may have been instances of this form, and that the fatality of the disease may not be so great as published facts suggest. But on this point only future observations can throw light. Chronic external meningitis which results from caries is only serious in the compression it exerts on the cord.

TREATMENT.—The local form alone affords an opportunity for effective treatment. The first and most important element is the treatment of the original cause of the disease, the caries, &c., to which the meningitis is secondary. Any accessible collection of pus should be opened, and if acute local symptoms, in a case of bone disease, suggest the passage of pus into the vertebral canal, the propriety of immediate trephining should be considered. Rest, counter-irritation to the spine, especially the actual cautery, sedatives to relieve the pain, and tonics are the most important measures in chronic cases. So far as the acute form is open to treatment, the measures suitable are those for the internal variety. In a subacute case, a free exit should be afforded to the pus. An opportunity for this very seldom presents itself, but may perhaps be furnished by sufficiently frequent examination more often than has hitherto been the case.

INTERNAL MENINGITIS.

Internal meningitis, inflammation beginning within the dura-matral sheath, may be either acute or chronic. The acute form usually commences in the pia mater and arachnoid; the chronic forms may begin in these membranes, or in the inner surface of the dura mater. Little is known practically of any separate affection of the arachnoid.

ACUTE INTERNAL MENINGITIS.

Internal meningitis, for the reason already mentioned, has been termed "leptomeningitis," but the acute form only remains limited to those membranes when of slight degree. In some cases the inner surface of the dura mater is also inflamed. Frequently the inflammation extends rapidly to the cord, and to such cases the term "meningo-myelitis" is often applied; in other cases, strange to say, no extension to the cord takes place. The inflammation may be either simple or purulent, or it may be secondary to tubercle. It also occurs in conjunction with cerebral meningitis in the epidemic form. Acute simple spinal meningitis is a rare disease.

The immediate causes that have been traced are in part such as have a local action; in part they are general morbid influences, such as give rise to other internal inflammations, and have been effective in causing meningitis in consequence either of some personal predisposition, or in consequence of a peculiarity in the agent, which led to a specific action on these structures.

(A) The local causes may be—(1) Injuries to the spine, severe or slight, from fracture to simple dislocation; concussion; surgical procedures, such as an operation on the vertebral column, or the puncture of the sac in spina bifida. (2) The exposure of the back to cold has been the apparent cause in rare cases; and still more rarely the prolonged exposure of the spine to the sun, "spinal insolation," has seemed to be effective. (3) Adjacent inflammation has sometimes caused internal meningitis, but in such cases external meningitis has of necessity been first produced, and the inflammation has passed through the dura mater. Hence, all the causes of acute external meningitis are also occasional causes of the internal form. But, as already stated, such extension through the sheath is very rare, and there is some doubt as to the precise nature of many of the cases in which it has been supposed to occur. We have seen that in many of the instances in which external meningitis has been ascribed to adjacent suppuration, the latter has been secondary, and the meningitis has been the result of a primary blood-state. When in such cases there has been both internal and external meningitis, it is probable that the former has not been due to actual extension through the dura mater, but that both have been the simultaneous result of a general cause.

(B) In a few cases acute internal meningitis is due to the extension of inflammation from the cerebral membranes. Such extension is rarely traced, however, except in two classes of cases:—(1) Those in which slight meningitis is found in the upper cervical region, as far as gravitation favours the descent of solid particles in the cerebro-spinal fluid as the patient lies in bed. The inflammation ceases when the spine becomes horizontal; for the most part symptoms are slight or absent in such cases. (2) In cases in which a general spinal meningitis has been secondary in point of time to inflammation of the cerebral membranes the affection has generally been due to some cause capable of acting upon both, such as the local presence of tubercles or a morbid blood-state of the character to be immediately mentioned.

(c) The most acute and severe forms of internal meningitis are due to a morbid blood-state, either septicæmia or a virus allied to that which causes the cerebro-spinal form. Purulent meningitis occurs in cases of surgical and puerperal septicæmia, both with and apart from a similar affection of the cerebral membranes. Acute inflammation, purulent when intense and prolonged, occurs also as an isolated malady, sometimes without apparent exciting cause, sometimes after exposure to cold. In such cases organisms have been found in the spinal fluids, analogous to those of the cerebro-spinal form, in connection with which the pathological relations will be more fully considered. It probably results from a blood-state not far removed from those which cause some other forms of internal inflammation, and which give rise to acute articular rheumatism. General exposure to cold must be assumed to cause so special an effect only through the agency of toxæmia, such as that which has been already considered as underlying the rheumatic form of acute polyneuritis. Such exposure has been apparently rendered effective in some instances by coincident menstruation, which may have produced a special susceptibility to the attack, or favorable conditions for the development of some organised virus, such as seems to be the essential factor in the causation of these forms. The exposure to cold that has been effective has presented all the variations that are met with in the case of other diseases thus produced, variations which are common to so many maladies that they can have no special relation to any one of them. Lastly, as a crucial example of this origin of the disease, is the important fact that it may follow, or occur in the course of certain acute specific diseases, as one consequence of the virus that produces them.

PATHOLOGICAL ANATOMY.—Internal meningitis is usually of wide extent, since the inflammation spreads readily in the loose tissue of the arachnoid. Probably also the movement of the cerebro-spinal fluid aids in the extension of local forms and in the descent of intracranial inflammation by conveying irritant material. In the case of a patient who had been in bed for some time the precise level at which such slight descending meningitis ceases may be that at which the vertebral column becomes horizontal. In the earliest stage the only change is congestion in the pia mater, which is reddened from vascularity, and may be dotted with ecchymoses. The inner surface of the dura mater, and the substance of the spinal cord, may be similarly congested. When the inflammation is further advanced, in the stage in which the condition most often comes under observation, the pia mater and arachnoid are opaque and thickened, and an "exudation" of inflammatory products, greyish yellow in tint, may cover the pia mater and occupy the meshes of the arachnoid, forming a layer over the cord. This exudation varies in its consistence, and may be semi-purulent in aspect; in the suppurative form the membranes are infiltrated with pus, which also covers their surface. The inner surface of the dura mater usually presents similar changes, and the inflammatory products may fill up the whole space between the dura and pia mater, thus connecting the two membranes and surrounding the nerve-roots (Fig. 90). The microscope shows abundant leucocytal elements, and the larger round and spindle cells that are common in all inflammatory products. The former corpuscles, which resemble, and are probably identical with pus-corpuscles, may be abundant, even when the exudation has not a distinctly purulent aspect; when it has, they constitute almost the whole of the material, lying among the fibres of the arachnoid (Fig. 91). The vessels are dilated, and their sheaths distended with cells. The spinal fluid is increased in quantity, and turbid from flocculi, or even purulent in aspect. The nerve-roots are covered with exudation, and are often swollen and reddened from invasion by the inflammation. But they do not always suffer, even in purulent meningitis, as Fig. 91 shows, in which a nerve-root is almost normal in aspect, and its sheath is unaffected, although it is surrounded by pus-cells. The spinal cord

FIG. 90.

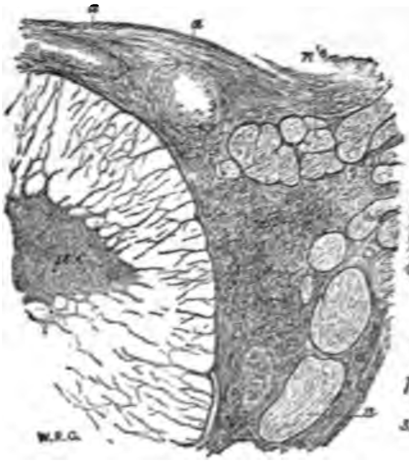

FIG. 90.—Purulent meningitis; portion of spinal cord and membranes; the space between the pia mater and dura mater is occupied by inflammatory products, pus, &c., in which the nerve-roots, *n n*, are embedded. *a a*. Cavities which had apparently been filled with liquid pus. From a case of septic origin, secondary to caries of the jaw.

FIG. 91.

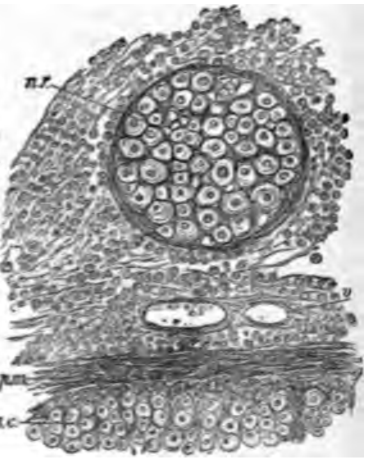

FIG. 91.—From the same; meshes of arachnoid infiltrated with pus-corpuscles. A nerve-root, *n. r.*, although surrounded by pus, is perfectly normal; and so also are the pia mater, *p. m.*, and the peripheral layer of the spinal cord, *s. c.*

is often invaded by the inflammation; it is then reddened, pale, and softened, and the microscope shows the tissue-changes common in other forms of myelitis. The change is always most marked in the periphery of the cord, and may occur in wedge-shaped areas, having the apex directed inwards, and coalescing at the surface. The peripheral arterioles come from the pia mater (see "Vessels"), and the continuation of their perivascular spaces with the lymphatic interstices of the membranes renders it easy to understand the invasion of the cord. Nevertheless, in some cases, especially of purulent meningitis, the pia mater itself is little affected, even when the arachnoid is filled with pus, and in these cases the spinal cord may be normal, as in the example shown in Fig. 90. If recovery takes place, the inflammatory products may undergo cicatricial changes, the membranes remaining opaque and adherent, and there may be a permanent excess of arachnoid fluid. The changes in the cord may lead to sclerosis, widely spread, or limited to certain spots, and from these secondary degenerations, ascending and descending, may develop.

The area affected varies in different cases; the membranes are involved in their whole extent in most acute forms. Usually the affection is greater on the posterior than on the anterior surface; probably on account of the influence of the recumbent posture, which determines the passage backwards of the lymphatic fluids containing material capable of exciting and increasing the inflammation.

In tubercular inflammation the amount of exudation is usually small, and it may be absent. It is often grey and gelatinous in appearance, and in it are scattered the greyish or whitish tubercular granulations. Similar granulations may usually be found in abundance upon the inner surface of the dura mater. Such grey granulations are often found upon the spinal membranes in cases of tuberculosis when there is no inflammation, even when the cerebral membranes are intensely inflamed. The arachnoid, especially that covering the cauda equina, may appear as if dusted over with grey particles, so abundant are the granulations.

In many cases, the signs of spinal meningitis are associated with those of inflammation of the membranes of the brain, especially of those about the base and posterior part of the brain. The continuity of the spinal and cerebral inflammation may be obvious or indistinct. In the latter case, the connecting inflammation has apparently been much slighter than that in the base of the brain or around the cord, so slight, indeed, as to have left no distinct traces, the inflammation having apparently extended in consequence of the passage of morbid material by the cerebro-spinal fluid. In cases of slight cerebral meningitis, in which the patient has been recumbent, signs of spinal meningitis, opacity of the arachnoid and its distension with slightly turbid fluid, may exist only in the cervical region, ceasing opposite the upper dorsal vertebræ, and thus extending as far as gravitation (the head being slightly raised on a pillow) favoured the descent of inflammatory products. But in the violent purulent cerebro-spinal meningitis, which is occasionally produced by a septic influence, pus around the cord is often continuous with that which bathes the base of the brain. In a case of this kind, in which the meningitis was secondary to acute double purulent otitis, Leyden found abundant active micrococci, very similar to those associated with erysipelas. In other acute cases, organisms have been found similar to those of pneumonia, and met with also in the epidemic cerebro-spinal form.

SYMPTOMS.—Slight pain in the back, and malaise, may precede the acute onset of the symptoms. This is usually marked by a rigor, by pyrexia, and by severe pain in the back. The latter varies in position, according to the locality of the inflammation, but is often felt along the whole extent of the spine. Pain also radiates along the distribution of the nerves, round the trunk or to the extremities. This excentric pain is paroxysmal and intense; sharp, darting, burning, or constricting. The pain in the back is usually constant, with exacerbations. It is often increased by movement, usually also by pressure, and by the application of a hot sponge to the skin. It is no doubt due to the irritation of the inflamed meninges, while the radiating pain is produced by the irritation of the sensory nerve-roots.

Muscular spasm usually comes on at the same time as the pain. It shows itself first in rigidity of the muscles of the back, most marked, if the inflammation is local, in the neighbourhood of the inflamed part. This rigidity is an important and characteristic symptom. It is often first observed in the neck, probably on account of the mobility of the part. When slight it may merely cause retraction of the head, or stiffness of the back, or may be so general and so severe as to cause opisthotonos, resembling that of tetanus. The spasm usually involves also other trunk-muscles, especially those of the abdomen, which become hard and cramped. The limbs also become rigid, and painful cramp-like spasms occur in them, especially on attempts to move. The spasm is probably partly due to the irritation of the motor nerve-roots, and is partly reflex, from the irritation of the sensory roots and the nerves of the pia mater. There is usually great hyperæsthesia of the skin to all forms of stimulation, and also increased sensitiveness of the muscles, especially in the lower limbs. Pressure on the arms may cause no uneasiness, while a similar pressure on the legs occasions great pain. Reflex action is usually increased at the beginning. Constipation is common, and so is retention of urine, in spite of irritable attempts to empty the bladder—the result, apparently, of spasm of its muscles, including the sphincter, which resists the action of the detrusor. Dyspnœa may result from the spasm of the thoracic muscles, and may be almost suffocating in its severity. The pulse may be frequent or retarded. The temperature is raised, sometimes, however, to only a slight degree. Cerebral symptoms, headache, delirium, coma, occur when inflammation has extended within the skull. The "Cheyne-Stokes breathing" may be present, from implication of the medulla. The function of the vaso-motor nerves (which leave the cord by many of the anterior roots) is also deranged; the dilatation of the vessels, that follows a stroke on the skin of the trunk, is excessive and prolonged (meningeal streak, *tache spinale*).

As the disease progresses, the symptoms of irritation give place to those of paralysis, which may be most marked where the rigidity was greatest. The limbs become relaxed, and feeble or powerless. Sensibility becomes lessened or lost. Reflex action disappears, the muscular power in the limbs becomes so reduced that the patient is scarcely able to move; the heart shares the universal prostration, and death may occur from asthenia, or from paralysis of the respiratory muscles. Towards the end there is sometimes considerable rise of temperature. In some cases the symptoms become less progressive and the disease passes into a less acute stage; the pains persist, the loss of power continues and may even slowly increase. Death may occur, after weeks of suffering, from the effects of bedsores or from secondary kidney disease, due to the retention of urine, and facilitated by trophic derangement. On the other hand, in slight cases, the signs of irritation may lessen and pass away, while those of structural damage, paralysis and anæsthesia, may remain, and to these may be added muscular atrophy and contractures, from the secondary consequences of the lesions of the nerve-roots. Such persistent symptoms vary much in extent and degree, according to the position and intensity of the morbid process. Ultimately the symptoms of damage to the cord may either slowly increase, in consequence of the spread of chronic myelitis set up by the acute mischief, or, on the other hand, the symptoms of the meningitis may pass entirely away.

The symptoms above described vary in their distribution, according to the position of the disease. When the membranes over the lumbar enlargement are chiefly affected, the pains, hyperæsthesia, and cramps are confined to the legs and loins. When the disease is in the dorsal region there may be similar hyperæsthesia and spasm in the legs, but the pain and cramp extend higher, and involve the trunk. If the cervical region is affected, the symptoms extend to the upper extremities, the dyspnœa may be great, and there is often difficulty in swallowing. The action of the heart is sometimes deranged, and contraction or dilatation of one or both pupils may occur. Extension to the brain is marked by vomiting, general headache, delirium, and paralysis of cranial nerves, of which the first to suffer are the spinal accessory and hypoglossal. If such symptoms preceded those of spinal meningitis, we may conclude that the inflammation commenced within the skull.

The symptoms vary somewhat according to the nature of the inflammation. In purulent meningitis, as already pointed out, the symptoms of irritation are sometimes very slight, apparently because there is little tendency to invade the nerve-roots. In the case from which Figs. 90 and 91 were taken, there were hardly any symptoms to suggest meningitis, and, although there was paraplegia, it was probably produced by the mere pressure of a large collection of pus on the spinal cord; there were no pains or spasm. The case was one of septic origin. It was apparently a pure arachnitis, and the tissue of the pia mater was able to resist invasion so completely as to protect the structures beneath it even from disturbance of function.

The duration of the acute symptoms varies from a day or two, in severe cases, which end in death, to two or three weeks, in cases of less severity, which may end in either death or recovery. The duration of the subacute and chronic symptoms that supervene is to be measured by months, and sometimes by years.

The symptoms of tubercular spinal meningitis resemble those which have been described, but are usually less intense. There is pain in the back and loins, with stiffness of the spine and retraction of the neck, so that it may be difficult to bend the head forwards. There are also variable rigidity, hyperæsthesia, and tingling in the limbs, followed by lessened sensibility and paraplegic weakness. These symptoms occasionally come on in the later stage of cerebral tubercular meningitis.

Diagnosis.—The diagnosis of the disease rests on the pain in the back, the retraction and rigidity of the neck and spine, the hyperæsthesia and spasm in the limbs, excited especially by attempts to move them, on the acute onset of the symptoms and on their association with pyrexia. This grouping of the symptoms is sufficiently characteristic, as is shown by the fact that it is extremely rare for them to be simulated, even remotely. The chief difficulty in actually acute cases is presented by the forms that run an almost latent course, especially by the secondary purulent form, which has so little tendency to invade the nerve structures, and a correspondingly slight tendency to derange their functions, and even to irritate them. In pure *myelitis*, on the other hand, pain in the back is absent or trifling; paralysis occurs early and is the leading symptom, and there is little or no spasm in the limbs in the early stage of the affection. Often, however, some meningitis occurs at the onset of acute myelitis, and then some pain in the back and slight rigidity in the limbs may precede or accompany the onset of the paralysis. In such cases the predominance of the meningeal or cord symptoms must determine the category in which the case is to be placed. *Meningeal hæmorrhage* most nearly resembles meningitis in its manifestations, and does so necessarily, because it produces inflammation; the distinction chiefly depends on the onset, and will be considered in the next section. *Hæmorrhage into the spinal cord* can scarcely be confused with inflammation of the membranes, because the only common symptom, pain in the back, is confined to a definite spot.

Tetanus is attended by rigidity of the back, and by spasm, and presents a closer initial resemblance to meningitis than most other affections with which it may be confounded; but there is no fever at the onset, trismus is an early and obtrusive symptom, and the paroxysms of muscular spasm are excited by peripheral impressions much more readily than in meningitis, in which they occur chiefly on attempts to move. Only the "rheumatic" form of tetanus would give rise to difficulty; the onset of the traumatic form after an injury should prevent any doubt. *Rheumatism of the muscles* may cause pain in the back on movement, and the resulting rigidity may prevent movement; but there is not the spontaneous pain which attends meningitis, nor is there distant spasm. Some difficulty may be caused by rheumatism of the cervical muscles in young children; this may lead to retraction of the head, and if it comes on acutely, after exposure to cold, considerable doubt as to its nature may at first be felt. But it remains stationary, and the freedom of the patient from spontaneous or radiating pains, and from any affection of the nerves, soon enables a reassuring opinion to be formed.

The diagnosis of the form of meningitis depends on the recognition of the cause of the inflammation. When spontaneous and acute the case is probably of the "primary" form, which depends on some toxic agency, probably often an organised virus, and is really a partial "epidemic" form. If it occurs under the conditions that give rise to surgical septicæmia, or after childbirth, purulent meningitis is almost certain, and the inflammation may be far more intense than the severity of the symptoms suggest. The diagnosis of tubercular spinal meningitis depends on the combination with cerebral meningitis, which usually precedes the spinal symptoms, and on the gradual and insidious onset. Indications of the tubercular or scrofulous diathesis are commonly present in the state of other organs, or to be ascertained from the family history of the patient.

PROGNOSIS.—The prognosis is grave in all cases. It is worse the more severe and acute the symptoms, the higher the temperature, and the sooner the symptoms of irritation give place to those of paralysis. It is worse also when the disease is due to serious lesions of the spine or to tuberculosis, than when due to cold, and worse in the so-called "spontaneous" cases than in those which result from traumatic causes. Recovery is more probable in middle life than in early or advanced age. The previous health of the patient also influences the prognosis. It must always be remembered that even if the patient survives the period of acute inflammation, serious permanent damage may remain.

TREATMENT.—Perfect rest and quiet are of the greatest importance throughout the course of the disease. All sounds should be, as far as possible, excluded; the light should be subdued, and all bodily movement and mental exertion as far as possible avoided. The vascular disturbance of inflammation is intensified by all functional excitement of the too irritable structures, and continuous freedom from such excitement, as far as it is possible to obtain the freedom, is of paramount importance. Every attack of spasm means greater vascular disturbance, and the circulatory derangement entails leucocytal infiltration and other structural consequences.

Although it is undesirable that the spine should be the lowest part of the body, yet, in acute meningitis, it is scarcely possible for the patient to lie in any other posture. The prone position interferes with respiration, and both it and a lateral posture entail, directly or indirectly, greater muscular exertion, and hence more frequent and more severe attacks of spasm, with secondary harm out of proportion to that which the posture could prevent. Here, as so often in therapeutics, skill is shown and success secured, if it is within reach, by knowing when to permit no compromise, and when to adopt one. The dorsal position must be permitted if in it the spasm is much less than in others. Dry, or, in robust patients, wet cupping, or leeching, along the spine may be employed at the onset, and be followed by the local application of cold in traumatic or hæmorrhagic cases, and of heat in others—the principles being the same as in myelitis. Counter-irritation, by blisters or repeated sinapisms, is more useful when the disease is subsiding than at the onset. In cases that are due to cold, free diaphoresis often does good; a hot air or vapour bath should be employed at the onset of the treatment. A warm bath may be followed by moist packing for several hours. The relief thus given is sometimes very great. The bowels should also be freely opened.

The only internal remedy which has been held, for long, in general repute, as capable of influencing the inflammatory process, is mercury. The confidence placed in it of old is not altogether unwarranted. It should be given until there is a slight affection of the gums (the only evidence that enough is in the system to act on the tissues), and inunction is unquestionably the best way of administration. We know that it must then enter the blood before it can escape by the bowel; we can regulate its dose as we need, for we estimate it by the influence on the gums; and by rubbing it in over the part affected we can combine some counter-irritant influence (as by an irritating agent added to the ointment used), and we necessarily secure the simultaneous action of the two agents, for the largest quantity of mercury in the blood will coincide with the most considerable reflex action on the vessels. Lastly, the disturbance of the stomach and bowels is certainly less than when their mucous membrane is chosen as the path into the body. The oleate of mercury may be rubbed in along the spine. Iodide of potassium seems to have little influence over acute inflammation.

It is necessary to give sedatives for the relief of the pain and spasm, if the application of cold does not suffice. Of these, morphia, given by the skin or the mouth, is the most effective, but vomiting from its use is a grave drawback; fortunately it does not readily occur under the circumstances. Sometimes inhalations of chloroform are necessary to relieve the suffering, and may be even more effective than morphia. In slight cases relief may be afforded by belladonna, or by atropia injected beneath the skin. Chloral, or chloral and bromide, or any of the milder sedatives, may be given if there is insomnia. Bromide alone, unfortunately, has very little power to lessen the spinal reflex action. Frequently, the spinal ice-bag is the most effective and least injurious.

When the disease has passed into a chronic stage, iodides have been thought useful. Counter-irritation may be employed freely with advantage. Tonics, iron, quinine, and even strychnia are beneficial. Warm baths, as those of Bath and Aix-les-Bains, seem sometimes of service, especially employed as hot douches to the spine. But the acute form is followed by a tendency to the slow subsidence of its effects, which makes the influence of such measures difficult to estimate. The local consequences, muscular atrophy, contracture, &c., need special local treatment by electricity, rubbing, and the like.

Epidemic cerebro-spinal meningitis is described in the section on diseases of the cerebral membranes.

CHRONIC INTERNAL MENINGITIS.

Chronic inflammation of the membranes of the cord, within the dura-matral sheath, is divided into two forms, according as it begins in, and chiefly affects, the dura mater, *chronic internal pachymeningitis*, or the pia mater and arachnoid, *chronic leptomeningitis*. Although these forms sometimes present distinct clinical and pathological features, they have, when of considerable degree, many characters in common; they own the same causes, and need the same treatment. Hence it is most convenient to describe them together as forms of internal meningitis. The condition termed *hæmatoma of the dura mater* depends on a form of hæmorrhagic inflammation.

Chronic meningitis is seldom fatal, and is rare as a general and primary malady. As such, therefore, our knowledge of it is still scanty, and the opinions formerly current have had to be largely curtailed. It was inferred that chronic symptoms, analogous to those which, when acute, are due to acute meningitis, were evidence of a chronic form. This was, therefore, assumed to be the cause of a group of symptoms, of which spasm is the prominent and dominant feature, which are now known to be due solely to a morbid state of the spinal cord itself, and to be consistent with a perfectly normal state of the membranes. Hence "chronic meningitis," as it was recognised twenty years ago, as a "clinical entity," has ceased to exist, or rather has passed into the pathological conception of "primary lateral sclerosis." The definite knowledge we now have of general primary chronic meningitis is limited. The important fact regarding it is that its symptoms differ from those of the acute form more widely, perhaps, than do those of any other chronic inflammation. Yet this difference depends on a comparatively small element—on the fact that only acute inflammation causes acute irritation of the motor and reflex structures. The chronic form may irritate the sensory nerve-roots, but its motor manifestations are chiefly the result of pressure, and even to this much of the pain may be due. The pressure may cause spasm, but it is a slow tonic contraction, wholly unlike that of the acute form. The difference in the effect of the two forms of inflammation suggests many problems that deserve investigation, but their consideration would be out of place here.

Causes.—Chronic internal meningitis, in every form, is most frequent in adult age, and, like acute inflammation, affects men more frequently than women. In its general and primary form, it has been thought to occur more readily in persons with neurotic heredity, although the influence of this is doubtful. Debilitating influences of various kinds predispose to the disease, and prolonged over-exertion has been thought sometimes to produce it. Among exciting causes the most important is severe and repeated exposure to cold. Traumatic lesions, concussion, &c., are occasional causes. It may result by extension from inflammation outside the dura mater and from chronic inflammation of the substance of the cord itself. But their influence in producing chronic internal inflammation (except as a sequel to the acute form) is a subject on which the opinions of the past have still to be subjected to careful comparison with ascertained facts. It is probable, however, that the condition does sometimes follow concussion of the spine that has no immediate or acute consequences, and the chronic stage into which an acute traumatic meningitis subsides may last so long as to throw its initial form into the shade, and may not only persist with independent pertinacity, but may extend without relation to the original seat. Thus we must distinguish as effects of injury, the local and the general forms. The latter are predisposed to by the influences that seem to facilitate the occurrence of the primary form, and in these cases the influence of the injury often seems to be trifling, and the existence of the morbid state is not always beyond doubt.

Local chronic meningitis may also result from any chronic disease either of the membranes themselves, the bones, or the spinal cord. These need not be enumerated in detail. It occurs in all cases of compression, and especially in all forms of chronic myelitis that involve the superficial layers of the cord.

Lastly, certain general morbid states are frequent causes. The first is alcoholism, which may give rise to general inflammation, involving both the pia arachnoid and the superficial layers of the cord in various degrees. A second and also common cause is syphilis, which may produce either pachymeningitis, surrounding the cord in the way presently to be described, or local leptomeningitis, irregular in position and in effects. Lastly, in very rare cases, tubercle has given rise to a chronic inflammation, chiefly of the inner surface of the ura mater. Hæmorrhagic pachymeningitis occurs especially in the nsane, but has been met with as a consequence of chronic alcoholism nd after injuries.

Pathological Anatomy.—In slight and moderate degree, there is nerely opacity and thickening of the membranes affected, sometimes rith distension of vessels or minute spots of extravasation. The pacity of the arachnoid may be such that the spinal cord cannot be een through it. The inner surface of the dura mater may be ranular when it is not otherwise changed. The spinal fluid is ncreased in quantity and is turbid. When the changes are greater n degree, the dura mater and pia mater may be connected together y a layer of inflammatory tissue of considerable thickness, so that it nay be impossible to say in which membrane the disease commenced. he microscope shows the ordinary elements which result from nflammation, cells of various kinds, many lymphoid and pus-like orpuscles, and distended vessels, often incrusted by similar cells. Frequently also the pia mater is transformed into a thick irregular layer of homogeneous tissue in which no distinct cell-elements can be

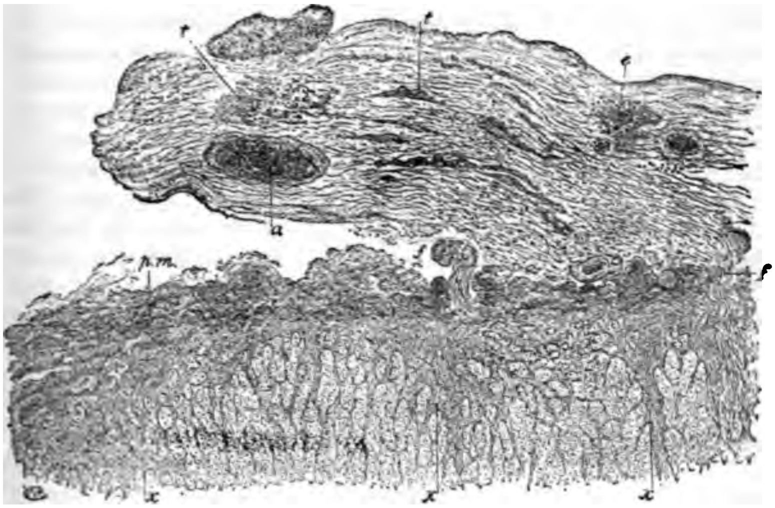

Fig. 92.—Chronic alcoholic meningitis. Section of edge of anterior column and of a large nerve-root; carmine preparation. *p. m.*, pia mater irregularly thickened and transformed into amorphous-looking tissue, from which wedge-shaped branching tracts (*x x*) extend into the white substance; *f. f.*, fasciculi of nerve-fibres entering the cord; *a.*, an artery in the nerve-root, enlarged and with thickened walls; *t. t.*, tracts of amorphous connective tissue; *e.*, a small extravasation.

perceived, and only faint indications of a fibrous structure (Fig. 92). The walls of its vessels may be greatly thickened by similar material.

The nerve-roots passing through the diseased membranes very seldom escape, as they may in the acute form, but are inflamed, reddened, and swollen in the active stage, and afterwards compressed and atrophied, if the amount of new tissue formed about them is considerable. The fibres suffer especially when the inflammation involves the dura mater, on account of the unyielding character of the fibrous sheaths which the nerves receive from that membrane. When the pia mater is thickened, scattered tracts of connective tissue may be seen in the substance of the nerve-roots, between the fibres (Fig. 92, *t*).

The spinal cord presents the same variations in the degree in which it suffers. It may be little affected, but is frequently damaged by the extension to it of the inflammation of the pia mater, which causes softening, vascularity, loss of distinction between grey and white substance, breaking down of nerve-elements, and infiltration by lymphoid and other inflammatory cells. Ultimately indurating tissue remains, constituting an irregular zone of peripheral sclerosis. From the thickened pia mater tracts of similar tissue may extend into the cord, becoming narrower as they pass inwards and sending out branching trabeculæ (Fig. 92, *s*). Between these tracts the nerve-fibres are more or less damaged, partly by the inflammation, and partly by the compression produced by the newly-formed tissue.

This peripheral sclerosis associated with chronic meningitis, especially in the neighbourhood of injuries, frequently damages tracts that undergo secondary ascending degeneration—post-median column, direct cerebellar tract, and ascending antero-lateral tract. The result is to produce a mixed lesion, the precise nature of which is not always easily distinguished and has certainly often been mistaken. The equivocal aspect of the process is probably increased by the fact that, as we have seen, secondary degeneration entails an interstitial process that is prone to assume an inflammatory character, especially when set up by an irritative lesion. Hence the peripheral sclerosis set up by the meningitis is apt to carry with it a certain amount of inflammation in the pia mater far beyond the region to which this would otherwise be limited. A similar thickening of the pia mater is, however, met with over areas of the cord that are the seat of other forms of sclerosis, both those that are systemic, as in tabes, and those that are random, as the insular form. It is probably secondary in all cases, but it formerly gave rise to some erroneous conceptions of the nature of such degenerations (see Locomotor Ataxy). A still more considerable implication of the pia mater is met with over regions that are the seat of focal chronic myelitis.

In the ordinary form of chronic meningitis, the membrane in which the inflammation begins may be much more affected than the other, although the latter is very rarely healthy, except in the slightest cases. Inflammation beginning in the pia mater and arachnoid is often extensive in range, and may be considerable or slight in degree. That which commences on the inner surface of the dura mater is frequently limited in extent, but is accompanied by the formation of a large amount of new tissue, and hence has been termed *hypertrophic internal pachymeningitis*. It constitutes a very important variety, giving rise to peculiar and grave symptoms which were first carefully studied by Charcot and Joffroy. It is most commonly the cervical region, but sometimes occurs at the lower part of the cord. On opening the spinal canal a fusiform tumour is seen, the outer surface of which is the unaltered outer surface of the dura mater, and on section the enlargement is seen to depend chiefly on a great thickening of the inner part of this membrane, sometimes amounting to a quarter of an inch; several layers of new tissue can often be distinguished. The pia mater may be normal, but is more commonly thickened, and it is often united by the thickened arachnoid to the tissue proceeding from the dura mater. The cord is compressed and commonly softened at the spot, and it presents signs of inflammation. The nerve-roots are also damaged and compressed by the newly-formed tissue. Sometimes at the spot most affected, the cord may be surrounded by a ring of new tissue of cartilaginous hardness. In other cases the thickening of the dura mater is slighter and more diffuse, affecting occasionally a wide extent of the membrane. In rare cases the membranes about the cauda equina may alone be affected—the dura and pia mater adherent, and the nerves united in a fibrous mass.

The white fibroid or cartilaginous plates found so often in the arachnoid after death are probably not connected with preceding inflammation. In most cases in which they are found, no symptoms have existed during life. It is said, however, that when they are numerous and extensive they may give rise to symptoms closely resembling those of chronic meningitis (Vulpian). But fibroid plates in the dura mater have been seen in a case in which previous symptoms suggested that they resulted from chronic inflammation (Jaccoud).

The rare condition termed *internal hæmorrhagic pachymeningitis*, or *hæmatoma of the spinal dura mater*, is similar to, and commonly associated with, that which affects the cranial membrane, and will be afterwards described. In this, a reddish-brown exudation covers the surface of the membrane, and is composed of fibrin and extravasated blood; the latter may be encysted in small cavities, or may be in various stages of transformation. The change commonly extends over a great part of the dura mater, and is apparently the result of hæmorrhage into inflammatory tissue.

The local form of syphilitic meningitis presents an irregular thickening of the pia mater and arachnoid, with the extensive production of new tissue in the affected region characteristic of this variety, so that it often resembles a diffuse growth as much as a chronic inflammation. The new tissue has a peculiar gluey aspect, and presents a tendency to caseous degeneration and fibroid change, so that scattered or coalescing cheesy spots are ultimately met with in tracts of mixed fibrous and gelatinous aspect, the later predominating in the more recent parts. This process of growth may attain such a development at some spot as to constitute a distinct "gumma," which may invade the substance of the cord, growing inwards from the pia mater. A similar process of cell-growth may be sometimes traced with the microscope into the substance of the cord, along the tracts of neuroglial tissue and peri-arterial spaces, when there is no invasion distinct to the naked eye.* A more limited affection of the walls of the arteries of the pia mater, extending into the cord, has also been thought to be syphilitic in nature; the whole thickness of the wall of the arterioles may be the seat of inflammatory thickening, in consequence of which the calibre of the vessels is narrowed.† There is, however, some reason to think that a similar affection of the vessels may occur apart from syphilis.‡

In the very rare chronic tubercular meningitis the inner surface of the dura mater is covered with a layer or layers of tubercles, in various stages of development, mingled with inflammatory products. The arachnoid may also contain degenerating tubercles, and the two membranes may be united together by the new formation. The condition occurs independently of bone disease.§

SYMPTOMS.—The symptoms of chronic internal meningitis, like those of the acute form, are due to the irritation of the membranes, to the damage to the nerve-roots, and to that of the cord. But the absence of acuteness in the process involves, as we have seen, a difference which produces a contrast, rather than a resemblance, in the manifestations of the two diseases; the spasm which dominates the aspect of acute meningitis is almost entirely absent in the chronic form. When this is eliminated, similar disturbances of function remain, but their relative subordination renders the similarity in the features of the two maladies unobtrusive. Still, the three pathological elements just mentioned occur in the chronic as well as in the acute form, and in different cases one or another of the corresponding sets of symptoms prodominate. Hence the varieties, while possessing many characters in common, may differ considerably in their clinical aspect. In some cases, we can infer with accuracy the special seat of the disease, but in many others we cannot carry our diagnosis beyond the conclusion that chronic internal meningitis exists, and are unable to say, even with probability, which membrane is primarily affected.

The common symptoms are these: Pain in the back, increased by movement, sometimes, however, amounting only to heavy dull discomfort, and accompanied by some stiffness of the back, and, when in the cervical region, by some retraction of the head. The pain is increased by pressure on the vertebral spines, and its increase by movement may cause a fixation of mobile parts, as the neck, in some abnormal position. The pain is apparently due to the irritation of the meningeal nerves, and the rigidity is to be regarded as a reflex effect of this irritation. But these are not the paroxysms of spasm, so conspicuous and so distressing in the acute form.

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* Gilbert and Lion, 'Arch. Gén.,' 1889.

† Schmaus, 'Deut. Arch. f. kl. Med.,' 1889.

‡ Hochhaus, 'Thesis,' Kiel, 1889.

§ Weiss, 'Wiener med. Wochensch.,' 1885.

More obtrusive, in most cases, are the radiating or excentric pains due to the irritation of the nerve-roots, and referred to the region in which the nerves are distributed, back of the head, neck, arms, thorax, abdomen, loins, or legs, according to the position of the disease. These pains are often very severe, sharp, darting, burning, or rheumatoid in character, paroxysmal in occurrence, and sometimes worse at night. They may be accompanied by a painful sense of constriction, and by various unpleasant sensations—numbness, tingling, formication, in the same areas. Hyperæsthesia may also exist, so that pain, sometimes thrilling in character, is produced by touching the skin, or by slight degrees of heat or cold, and a hot or cold sponge passed along the skin of the back may reveal the chief seat of the disease by the change of sensation at its level, as in the acute form. The hyperæsthesia may be more marked to one form of sensation than to another. Cutaneous eruptions, such as result from other nerve-lesions, have also been observed in association with the pains. In the same regions, muscular twitching, tremor, or even spasm, may result from the irritation of the motor root-fibres. These symptoms correspond in their level to the part of the membranes chiefly diseased. When the lumbar membranes are affected, the radiating pains are felt in the legs; but when the disease is higher up, the legs may merely feel heavy, and be the seat of slight abnormal sensations.

After a time, weeks or months, the pains persisting or ceasing, certain paralytic symptoms manifest themselves in the regions in which the severe pains were felt, and are dependent on further damage to the nerve-roots. Sensation becomes lessened, or even lost, in certain areas, to touch, or pain, or both. The muscles in the same region become weak, and waste; usually with the reaction of degeneration for a time, and ultimate loss of all irritability. The wasting often affects the muscles irregularly, from the unequal damage to the roots and the fibres of single roots: entire groups sometimes atrophy from the complete interruption of the roots of one or more segments. Reflex action in these parts is lost. In the trunk this loss of reflex action, with anæsthesia, may be the chief local symptom, and is of much diagnostic importance. As the disease progresses, the cord itself suffers, by compression, or by extension to it of the inflammation. The parts below the seat of the disease then become paralysed, the legs are weak and the seat of dull heavy pain; reflex action in them may be increased, while their muscular nutrition continues good, unless the lumbar membranes are diseased, or the myelitis descends the cord. Defect of co-ordination in the legs is not uncommon, and is probably due to the implication of the fibres from the muscles in the posterior columns and cerebellar tracts. Power over the sphincters may be lost and bedsores form, but chiefly in cases in which the disease is severe over the lumbar region or cauda equina.

When the inflammation affects chiefly the pia mater and arachnoid (*chronic leptomeningitis*) the symptoms of meningeal irritation are conspicuous,—vertebral pain and rigidity of the back, with cutaneous hyperæsthesia, and the radiating pains may also be considerable. These symptoms are especially marked in cases of subchronic meningitis, such as are sometimes due to alcoholism. The local paralytic symptoms—anæsthesia and muscular wasting—may be absent in these cases, or may only come on in slight degree after the other symptoms have lasted for a considerable time. On the other hand, weakness and pains in the legs, from affection of the cord itself, may be early symptoms.

When the inflammation begins in the dura mater (*internal pachymeningitis*) the symptoms of spinal irritation, pain and stiffness of the back, although occasionally present, are much less prominent symptoms than are those which depend on the irritation of, and damage to, the nerve-roots. The radiating pains are very severe, and are often the earliest symptom. At a later stage the muscular weakness, followed or accompanied by wasting, chiefly attracts attention, and the wasting may be so great that the disease is mistaken for progressive muscular atrophy. This is especially the case when the lesion is in the cervical region, the "cervical hypertrophic pachymeningitis" of Charcot and Joffroy. In this affection, pains in the back of the head, neck, shoulders, and arms, often accompanied with a painful sense of constriction, precede the muscular atrophy. The wasting may involve many muscles, but usually those supplied by the radial nerve are less affected than the others; the escape of the long extensors of the wrist and fingers, while the flexors of the wrist and fingers and the interossei atrophy, leads to a peculiar deformity; the unopposed muscles cause persistent over-extension of the wrist; the phalangeal joints are flexed and the metacarpo-phalangeal joints are extended, but from the position of the wrist they are not over-extended, and the hand thus differs from the claw-like hand (p. 30). With this muscular wasting in the arms there is paraplegic weakness, from the pressure on the cord. In rare cases the disease affects the membranes over the lumbar enlargement or cauda equina, and then the legs are the seat of the pains, paralysis, and atrophy already described, and the sphincters are early paralysed.

If the pachymeningitis is confined to the dorsal region, the root-symptoms are limited to the trunk. In such a case anæsthesia over the region supplied by the dorsal nerves was associated with paraplegia from compression of the cord. In another, areas of anæsthesia, with severe pains, existed over the legs, trunk, neck, and back of the head, without any indication of damage to the cord itself. These examples illustrate the great variations presented by different cases, owing to the different distribution of the disease. An important characteristic of this form is the fact that it involves the membranes equally around the cord, and so gives rise to bilateral symptoms.

Chronic syphilitic meningitis may cause symptoms resembling those of any other variety of chronic inflammation, even the hypertrophic pachymeningitis being sometimes due to this cause. But its most frequent and special characteristic is the focal character of the symptoms, and their tendency to be associated with those of partial and local damage to the spinal cord itself. This is in consequence of the tendency of local tissue-formation to be associated with the inflammation. Hence, pains of irregular distribution on one side are usually soon followed by impairment of sensibility, and often by weakness in the corresponding leg.

In hæmorrhagic pachymeningitis the symptoms are those of a slight chronic inflammation, without any distinctive features that can be referred to the hæmorrhagic infiltration.

DIAGNOSIS.—The diseases with which chronic internal meningitis is liable to be confounded, are different according as the symptoms of irritation, of damage to the cord, or of compression of the nerve-roots, predominate. By far the most important diagnostic symptoms are those that depend on the damage to the nerve-roots—their early irritation and later structural damage.

The spinal pain and stiffness may be mistaken for simple rachialgia or "spinal irritation," but in this condition the whole spine is tender, several separate points are much more tender than the rest, and there are not the radiating pains or paralytic symptoms of meningitis. Pain in the back is related in greater degree to posture and exertion, and it has a tendency to pass from the neck to the occiput. The patients have usually suffered from neuralgia in other situations, which may alternate with that in the back.

Peripheral pains, especially about the trunk and legs and shoulders, are prominent symptoms in some cases of muscular rheumatism or rheumatic neuralgia. In these, however, there are not the pain and tenderness of the spinal column; and although there may be apparent increased sensitiveness in the region of the pains, it is of the deeper structures and not of the skin, while the relation of the pain to movement and muscular action is a conspicuous feature.

Radicular neuritis is probably associated in many cases with some affection of the adjacent membranes, but meningeal symptoms are altogether subordinate; the symptoms are confined to the region supplied by the nerve-roots, and spinal pain is absent.

The muscular wasting may be mistaken for atrophy due to degeneration or subacute inflammation of the anterior cornua of the cord (progressive muscular atrophy, subacute polio-myelitis). The cervical hypertrophic pachymeningitis bears special resemblance to this disease, since the wasting in the arms is often associated with paraplegic symptoms in the legs. The distinction is that the atrophy, in the meningeal form, is preceded by severe pains, and often accompanied by areas of hyperæsthesia or anæsthesia, and is random in distribution. In chronic affections of the anterior cornua both pains and anæsthesia are usually absent, and the wasting often affects the muscles in a certain order, which will be described in the account of the disease. Thus, in a man who was supposed to have cornual disease on account of wasting of some muscles of both legs, irregularly distributed, a history of severe sharp pains, and the discovery of a patch of anæsthesia on one leg, the presence of a burning pain round one side of the trunk, and the irregular distribution of the wasting, led to a diagnosis of chronic meningitis damaging the nerve-roots; there was a history of syphilis, and appropriate treatment completely removed the symptoms. The pains also constitute a distinction from another affection which presents somewhat similar symptoms—syringomyelia. Chronic disseminated myelitis may cause muscular wasting, but does not cause the pain in the back, or the rigidity of the spine, which occur in meningitis, and the paralysis from damage to the cord is the leading symptom. It must be remembered that the two conditions of meningitis and myelitis are often combined, and the fact has to be recognised in our diagnosis.

Some authorities believe that the symptoms of "ataxic paraplegia," which result from combined lateral and posterior sclerosis, are often produced by the agency of chronic meningitis, invading the peripheral layers of the cord. It is not probable, however, that this mechanism is operative in the cases in which considerable paraplegia shows that the lateral pyramidal tracts are extensively involved. The diagnostic problem is considered in the chapter on this disease.

When the meningitis is lumbar, the pains in the legs may resemble some of those that occur in locomotor ataxy, and knee-jerk may be lost in each disease; but the loss in meningitis is associated with muscular weakness and wasting, there is not the degree of ataxy common in tabes, and true "lightning pains" are not met with in meningitis. The greatest resemblance to chronic meningitis is presented by some cases of tabes in which the cord disease reaches a greater degree in the dorsal than in the lumbar region. There are then pains and anæsthesia in the trunk, and the atrophy in the legs may be little marked. In meningitis, if the membranes over the lumbar enlargement are normal, the knee-jerk is preserved and often increased, while it is lost in tabes. The anæsthesia and loss of the trunk reflexes are more general and uniform in tabes than in meningitis.

Caries of the spinal bones produces many of its symptoms by the agency of chronic meningitis, which always results from it. Hence the diagnosis depends on the recognition of the presence of the disease of the bone, in addition to that of the membranes.

Multiple neuritis, in the paralytic variety, presents nerve-symptoms of the same character as those of chronic meningitis, but the special symmetry and peculiar distribution of the atrophic palsy, the prominence of limb-tenderness, and the absence of cord symptoms, preclude such similarity as can cause actual confusion between the two diseases.

Prognosis.—The differences between the forms of chronic meningitis, and between the degrees of the same forms, are so vast as to render the prognosis of the disease a matter of individual consideration in every case, to be estimated independently, from the pathological characters. The severe degrees of the affection are attended with danger to life, and even the slighter forms may entail serious consequences, since many effects of the disease, especially the damage to the spinal cord itself, tend to increase by the myelitic tendency which is set up. The neuralgic pains, which result from the damage to the nerve-roots, are extremely obstinate. But in many cases the symptoms ultimately pass away—the irritation ceases and the compressed structures slowly regain their function. The prognosis is most favorable in the cases which result from injury and from syphilis. But in all cases, even those of syphilitic origin, in which there is reason to infer considerable formation of new tissue, the prognosis must be cautious, since the cicatricial contraction of this tissue may perpetuate the damage to cord and nerves which its pressure has produced. The prognosis is better, in other cases, in proportion to the general health of the patient, to the moderate degree of the mischief, and to the extent to which its causes are under control.

Treatment.—Rest is essential; in severe cases it should be absolute, and in every case movements which increase pain should be avoided. The pain is the expression of irritation, which tends to maintain the morbid process. Measures, previously ineffective, will often be followed by improvement if rest is added. Posture is also of great importance. The prone couch, impracticable in acute meningitis, can often be employed in chronic cases with advantage, to lessen mechanical congestion of the spine. Warm baths and diaphoretic baths have been recommended as useful, especially in subacute cases. Next in importance to rest and posture is counter-irritation. Repeated sinapisms or stimulating liniments may be used if mild counter-irritation only is required, but in most cases greater good will result from more energetic means, blisters, the thermic hammer, or the actual cautery. The last is especially recommended by Joffroy for the hypertrophic pachymeningitis. It is probable that posture favours the beneficial influence of counter-irritation in a very important degree.

Sedatives are usually needed to relieve the pain—morphia, chloral, Indian hemp, injections of morphia or cocaine, or sedative liniments of chloroform, belladonna, &c. In employing sedatives it must be remembered that they will usually be needed, in chronic cases, for many months, and after a time, by the artificial need they create, they may apparently keep up the pain. It is therefore well, if possible, to alternate two or more sedatives, to make cocaine take the place of morphia as much as possible, and to lessen the dose from time to time, tentatively, and without the knowledge of the patient.

Iodide of potassium and mercury are the agents that have most influence over the morbid process itself. Both are effective in syphilitic cases, but in other forms of the disease mercury has far more influence than iodide; it may be rubbed in over the spine, as recommended in the acute form. The ointment and oleate may be rendered counter-irritant, if necessary, by the addition of cantharides ointment. In many cases, tonics, as iron, quinine, and cod-liver oil, are needed. The muscular wasting which results from damage to the nerve-roots requires treatment by galvanism, rubbing, and by passive movement, to prevent contractures. If hæmorrhagic pachymeningitis is suspected, ergotine may be used in addition to the measures suited for the ordinary form. Treatment appears, however, to exert little influence upon it.

Other measures are the same as have been described for the acute form.

HÆMORRHAGE INTO THE SPINAL MEMBRANES; HÆMATORACHIS.

Spinal hæmorrhage may be outside the dura mater, between it and the bones (*extra-meningeal*), or within the dura mater (*intra-meningeal*). In the latter situation the extravasation may be *sub-dural*, between the dura mater and arachnoid, or it may be *sub-arachnoid*, between this and the pia mater. Hæmorrhage outside the dura mater is more common than within it.

Causes.—Meningeal hæmorrhage may occur at all ages. It is met with in newly-born children, but in them is due to rupture of vessels during birth, and the blood often merely descends into the spinal membranes from those of the brain. Spontaneous hæmorrhage is unknown in early childhood, but occurs at all other ages, and is more common in men than in women. Of the immediate causes, injury is the most frequent, fractures of the spinal column, blows or falls on the back which do not fracture the vertebræ, and even falls on the feet or buttocks. It is occasionally found after death from severe convulsions, epilepsy, puerperal eclampsia (in which an altered state of the blood may assist), chorea, strychnine poisoning, and tetanus. In some instances no symptoms were observed during life which could be referred to the hæmorrhage, and it is probable that the extravasation occurred only during the last moments of life. Hence it seems likely that muscular spasm, resulting from a meningeal hæmorrhage, may sometimes have been looked on as primary and independent—the cause of the hæmorrhage when it was really the effect thereof. This seems certain in many cases of tetanoid spasm in newly-born children—in most cases, indeed, in which such spasm occurs almost immediately after birth and is associated with meningeal hæmorrhage. Such cases should be separated from those to which the term *tetanus neonatorum* is applied. They are further considered in the account of hæmorrhage into the cerebral membranes in vol. ii. Severe and prolonged muscular exertion has been, in a few cases, the apparent cause of spontaneous hæmorrhage. It also occurs in some diseases in which there is a hæmorrhagic tendency, such as purpuric states, and the hæmorrhagic forms of some acute specific diseases, smallpox, yellow fever, &c., and very rarely in typhoid fever, apart from any hæmorrhage elsewhere. In most of these cases its occurrence has not been suspected until the post-mortem examination revealed it, and it probably occurs generally during the last moments of life. A rare cause is the bursting of an aortic aneurism into the spinal canal, after erosion of the bodies of the vertebræ, the blood being effused outside the dura mater. Hæmorrhage within the dura mater has also resulted from the rupture of an aneurism of a vertebral artery. Blood may descend into the spinal membranes from the cranium, in cases in which a cerebral extravasation, especially about the pons, escapes into the meninges; this may occur in adults as well as in the meningeal hæmorrhage that is produced during birth. Lastly, in cases of intense meningeal inflammation, ecchymoses, and sometimes considerable extravasations, have been found on both sides of the dura mater or in the pia mater.

PATHOLOGICAL ANATOMY.—*Extra-dural* hæmorrhage comes from the plexus of large veins which lie between the dura mater and the bone. It is usually not of large extent, and the blood collects chiefly on the posterior aspect, where, in the recumbent posture, gravitation favours accumulation, and the space between the membrane and the bone is greater than in front. But sometimes the extravasation is very extensive, covering a large part of the dura mater, and in such cases it may extend through the intervertebral foramina, along the nerves. Hæmorrhage is more common in the cervical region than elsewhere, but may occur in any part. The blood is usually coagulated, wholly or partially. The dura mater is blood-stained, sometimes even through to the inner surface. The spinal cord may be compressed, but the amount of blood is not often sufficient for this.

It is easy to fall into grave error regarding the presence of meningeal hæmorrhage; when a body has been lying on the back, the veins outside the dura mater become distended with blood, and this escapes when their walls are divided in opening the spinal canal. Care must be taken to avoid the error, on the one hand, of regarding the blood thus escaping as an ante-mortem extravasation, and, on the other, of overlooking a hæmorrhage which actually exists. In all cases of acute spinal disease, in which a post-mortem examination is to be made, the body should at first lie face downwards.

Intra-meningeal hæmorrhage.—An extravasation into the sac of the dura mater (subdural hæmorrhage) may be small in quantity, or may fill the whole cavity. When small it may pass from one part to another. In subarachnoid hæmorrhage, the blood comes usually from the vessels of the pia mater; it may surround the cord for an inch or two, or may fill the whole subarachnoid cavity. Such extensive effusions are rare, except when the blood descends into the arachnoid from the cerebral membranes. Blood effused into the spinal arachnoid has been known to ascend as high as the pons, and even to break through the valve of Vieussens and get into the cerebral ventricles.* Simultaneous cerebral and spinal hæmorrhages have been known to occur from a common and powerful cause, as, for instance, in a case of puerperal eclampsia (Charrier).

In meningeal hæmorrhage, cerebral as well as spinal, the spinal fluid is often blood-stained, and thus spinal hæmorrhage may be thought to be more considerable than it really is. The spinal cord is often discoloured and compressed, and is especially damaged when the hæmorrhage is beneath the arachnoid. In extensive extra-meningeal hæmorrhage, the subarachnoid fluid may be driven away from the compressed part, and may distend the arachnoid beyond the area of compression, the limit of which is thus marked. In cases which have lasted more than a few days there are usually signs of meningitis, set up by the irritation of the blood.

Symptoms.—As already stated, slight meningeal hæmorrhage is sometimes found post mortem in death from convulsive diseases when no symptoms of it were observed during life, and in these cases it is probable that the extravasation occurs during the lethal convulsions. In the cases in which hæmorrhage causes symptoms, these are, for the most part, those of meningeal irritation, and thus bear some resemblance to the symptoms of meningitis. They differ in the suddenness and violence of their onset, to which exceptions are extremely rare. These symptoms are nearly the same whether the hæmorrhage is outside or inside the dura mater.

The first indication of the lesion is usually sudden and violent pain in the back, corresponding in position to the seat of the hæmorrhage, but generally felt along a considerable extent of the back, and often severe in the loins. This pain in the back is usually accompanied by pain along the course of the nerves passing through the membrane near the extravasation, darting or burning pains, often of great intensity; they are paroxysmal in character, and between the pains there may be various abnormal sensations, tingling, &c., referred to the same parts, and accompanied by hyperæsthesia. Muscular spasm usually coincides with the pain, and involves partly the vertebral muscles, causing rigidity of the spine or actual opisthotonos, partly the muscles supplied by the nerves in which the pain is felt, partly the muscles supplied from the cord below the seat of the hæmorrhage. The convulsive movements are sometimes general. Intense pain in the back and general convulsion have been known to be the only symptoms. Occasionally there is persistent contraction of muscles, and there is usually spasmodic retention of urine. These symptoms of irritation are no doubt due in part to the irritation of the membranes (causing the vertebral pain and reflex spasm), and partly to the irritation of the nerve-roots, motor and sensory. Paralytic symptoms quickly follow—weakness and lessened sensitiveness in the limbs below the lesion. In some cases there is, at last, complete loss of motion and sensation in the lower limbs, but such absolute paralysis is not common. Loss of power at the onset of the symptoms usually indicates either simultaneous hæmorrhage into the cord or the effusion of a very large amount of blood. It occurs, for instance, when an aneurism bursts into the spinal canal.

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* As in an apparently conclusive case reported by Lepretre, 'Arch. Gén. de Méd.,' xxii, p. 331.

The symptoms necessarily differ in situation according to the seat of the disease. When hæmorrhage is in the cervical region (a frequent seat), the pains are felt in the neck and arms, and the rigidity may cause absolute immobility of the neck, while dysphagia, interference with respiration, and dilatation of the pupils may be added to the other paralytic symptoms. When it is in the dorsal region, intense pain encircles the chest or abdomen. If in the lumbar region, the pain is felt in the legs, and there are early paraplegic symptoms, with loss of reflex action in the legs, and the paralytic incontinence of urine and fæces described on p. 225.

As a rule, in spinal hæmorrhage, the cerebral functions are unaffected; the unfortunate patient is conscious of all his sufferings from first to last. Occasionally, however, consciousness is lost for a short time, apparently from shock, and delirium or coma may come on, either as an indirect effect of the spinal lesion on the brain, or in consequence of a sudden increase in the intra-cranial pressure, due to the displacement upwards of the cerebro-spinal fluid. But in most cases in which cerebral symptoms coexist with those of spinal hæmorrhage, the former have been due to simultaneous intra-cranial disease; this is the case when the blood found in the spinal canal comes from within the skull, as in most cases of meningeal hæmorrhage in newly-born children (see vol. ii). In very rare cases the symptoms of spinal hæmorrhage have come on insidiously, without pain, as in a case in which extensive hæmorrhage outside the dura mater in the cervical and upper dorsal region, in a girl of fourteen, caused only very gradual weakness in the arms, and difficulty of breathing, from which she died at the end of a fortnight.* Such a case illustrates the almost universal law that there is hardly any "characteristic" symptom of a disease that is not sometimes absent, and that the absence of such symptoms has incomparable less significance than their presence.

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* R. Jackson, 'Lancet,' 1869, p. 5.

The symptoms of meningeal hæmorrhage, in acute cases, usually reach their height in two or three hours, but sometimes not for a few days. Death may occur when the effects are fully developed; or, after the symptoms have reached their height, they may decline, to be renewed, a day or two later, by secondary meningitis. This is accompanied by some pyrexia, and may cause death; but if moderate, the symptoms of inflammation last a week or ten days, and then, in favorable cases, permanent improvement commences.

The period of the disease at which death is most common is a few hours after the onset of the symptoms. It may be due to exhaustion, in consequence of the violence of the pain and spasm, but is more often caused by the interference with respiration that results from the conjoined effect of the palsy and spasm.

DIAGNOSIS.—In cases in which definite symptoms are produced, the diagnosis rests on the combination of sudden local pain in the back, with the other evidence of irritation of the membranes, nerve-roots, and cord, above enumerated. When the hæmorrhage is of traumatic origin, the fact that the symptoms rapidly followed an injury facilitates the diagnosis. In the extremely rare cases in which the symptoms come on insidiously, without pain, the diagnosis of the exact nature of the disease is scarcely possible.

In hæmorrhage into the substance of the cord, vertebral pain is much more frequently absent than in meningeal hæmorrhage, and when it occurs is less intense and does not spread. The symptoms of injury to the cord itself are prominent from the commencement; there is sudden paralysis, it may be at first partial, and rapidly increasing; even if there is some spinal pain at the onset, the loss of power is soon recognised, not being concealed either by the severity of the pain or by spasm. In meningeal hæmorrhage, severe pain and symptoms of irritation usually precede considerable paralysis. In cases which recover, the paralytic symptoms are more persistent when the hæmorrhage is into the substance of the cord. But hæmorrhage into the cord often breaks through into the membranes, and the symptoms of both lesions are then combined.

Meningitis is distinguished by the more gradual onset of the symptoms and by the presence of fever from the first; the fact that inflammation results from hæmorrhage must be kept in mind. In myelitis, pain is commonly absent, and the symptoms of irritation are insignificant. The diagnosis from tetanus depends on the absence of trismus, on the presence of severe spinal pains, and on the sudden onset. In newly-born children the diagnosis is supposed to be difficult, but during the first few hours or days the tetanoid symptoms which sometimes occur are the result of meningeal hæmorrhage and injury to the motor cortex.

One case of spinal hæmorrhage has been recorded in which the symptoms very closely simulated those of strychnine poisoning. Violent paroxysms of muscular spasm, with intense general pain, but without spinal pain, followed by death in two hours, were apparently due to an extensive hæmorrhage into the sac of the dura mater.* Analysis revealed no strychnia in the stomach. In such a case the diagnosis would have to be guided as much by circumstantial evidence as by the symptoms, and even the post-mortem discovery of hæmorrhage could only be received as evidence of the cause of the convulsion if no other were found.

Prognosis.—In all severe cases, death in a few hours is to be feared. The prognosis only becomes less grave when the symptoms have distinctly reached their height, although there is still some danger until the period of secondary inflammation is over. Paralytic symptoms often persist for some time, but even these, in the end, may pass away. At the onset, the prognosis must be governed by the rapidity with which the symptoms develop and by the seat of the disease. Hæmorrhage in the cervical region is especially serious, because the mechanism for breathing is directly involved.

Treatment.—The first point to secure is absolute rest. Even passive movement should be avoided. Posture is also of most urgent importance. It should be on the face or side, and not on the back, so that the spine may not be the lowest part. The prone position for a few hours adds greatly to the chances of less damage. Venesection has been employed in strong individuals, with the object of arresting hæmorrhage by rapidly lowering the blood-pressure. Leeches or wet cupping to the spine, or leeches to the anus, have also been recommended in cases in which venesection is unadvisable. It is probable that the local abstraction of blood does more in this than in most internal hæmorrhages, on account of the communication between the venous plexuses and the veins in the retro-vertebral tissues; and it is probable that the wisest treatment at the onset would be scarification beside the spine, opposite the seat of pain, the blood being allowed to flow freely. This may be followed by the application of ice and by the administration of ergotine by the skin or mouth. Afterwards the bowels should be freely opened. Sedatives are usually required to relieve the pain. The stage of meningitis must be treated on the principles laid down for the management of the acute form of that disease. The residual palsy requires treatment by electricity, &c., in the same manner as the consequences of meningitis.

In extra-meningeal hæmorrhage it seems justifiable to remove blood that is compressing the cord, by opening the canal and perhaps even washing out the canal with an astringent and antiseptic solution. Even when the blood is within the dura mater, the operation seems to deserve consideration if life is threatened.

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* Dixon, 'Lancet,' 1879, p. 333.

DISEASES OF THE SPINAL CORD.

ANÆMIA AND HYPERÆMIA OF THE SPINAL CORD.

The condition of the vessels of the spinal cord after death, their fulness or emptiness, affords no indication whatever of their state during life. Inferences as to the ante-mortem state, drawn from the post-mortem condition, are altogether erroneous, with the rare exception of local change, in which the vascular condition of one part differs from the rest. Practically, such local variation occurs only as part of a local lesion, as, for instance, in the hyperæmia that attends inflammation, and the anæmia that results from pressure. Hence the occurrence of variations in the state of the vessels of the cord, and the effects that such variations may produce, are matters of inference from symptoms observed during life—symptoms that are, in themselves, open to various interpretations. Where the ground is barren of facts, theory is always luxuriant. Anæmia or congestion of the cord affords a ready explanation of symptoms the cause of which is unknown, and it is scarcely surprising, therefore, that such an explanation has been often given. But the extent to which the conditions have been invoked transcends any measure of justification, and surprise cannot but be felt at the absolute confidence and precision of detail with which these states have been assumed to exist, when there has been no tittle of definite evidence. Positive assertions always receive some credence, however unwarranted the assertions may be, and positions incapable of proof are sometimes also incapable of disproof. It would be a futile and useless task to attempt to refute in detail the various statements that have been made regarding the influence of anæmia and hyperæmia of the spinal cord. It will be sufficient to point out briefly what may be reasonably surmised regarding these morbid states.

ANÆMIA OF THE CORD.

Permanent diminution in the blood-supply to considerable areas of the spinal cord must be a result of the general narrowing of the arteries that has been met with in some cases of chronic meningitis; but it is then associated with structural changes in the organ, the effects of which obscure any symptoms that can be ascribed to the anæmia. Nothing is practically known of the special consequences of this condition. The same statement may be made of the arrest of the blood-supply to limited areas, although it may safely be asserted that loss of function of the part must be a necessary consequence. This has been also demonstrated by experiment. Suddenly produced, anæmia at once abolishes function and quickly causes necrosis of the nerve-elements—some of which, however, break up less rapidly than others, and retain, for a longer time, the power of resuming function if the blood-supply is restored. It is possible that such local arrest of the circulation is the initial process in some organic lesions of the cord, but the fact has still to be demonstrated. The question of the occurrence of embolism is referred to in a separate section.

Transient diminution in the blood-supply may conceivably be the result of imperfect embolism, but has not been proved to have this origin. It is sometimes supposed to be due to arterial spasm, dependent on the vaso-motor nerves. This has been assumed as an explanation of certain passing symptoms occasionally complained of, loss of power in the legs, and sensory disturbance, tingling, "pins and needles," &c., in the legs and arms. It may possibly also be the cause of the tetanoid cramp and tingling in the hands with which patients sometimes wake up during the night. The theory is tenable, but it is also conceivable that such symptoms are due to transient functional states of the nerve-cells of the cord. A functional derangement of these cells must be the immediate cause of the symptoms, whether such derangement is primary or is produced by spasm of the vessels. The opinion that the "nocturnal tetany" (as it may be called) has the latter origin is supported by the fact that the occurrence of the symptom may be prevented by a small dose of digitalis, taken at bedtime.

The curious symptom known as "intermitting lameness" (which would be better termed "paroxysmal lameness") is supposed to depend on temporary diminution of the blood-supply consequent on arterial degeneration, but it is not known whether the seat of this is the spinal cord or the brain. The character of the symptom suggests the former, but its associations the latter, and it is not known that the arteries of the cord are ever the seat of atheroma in the cases in which this condition causes the cerebral symptom commonly associated with it, "dyslexia," a peculiar intermitting difficulty in reading which will be described in vol. ii. The lameness consists in a sudden weakness and stiffness of one leg, accompanied with tingling and blunting of sensibility, and of pain on an attempt to use it. The symptoms are increased by an effort to continue walking, and pass away after a rest of ten minutes or so, usually returning, on renewed exertion, again and again. The difficulty only occurs during exertion. It is familiar to veterinary surgeons as occurring in horses. The dyslexia with which it may be associated is of grave significance, as the cerebral cause seems always to have a strongly progressive character.

In *general anæmia*, such as occurs in chlorosis and pernicious anæmia, the nutrition and function of all organs suffer, and the spinal cord shares the general state. The readiness with which fatigue of the legs is induced in such conditions may be due to the spinal cord as well as to the muscles. The legs often ache, and are sometimes the seat of various disordered sensations. It is probable that these are the result of the impaired nutrition of the nerve-elements, in consequence of which their functions are readily deranged. In some patients graver symptoms occur,—weakness of the legs, sometimes of the arms also, which may increase slowly to complete paralysis. Sensation and the sphincters are usually unaffected. The nature of these cases is uncertain. It is probable that many are cases of what is called hysterical paraplegia. Others may be due to organic changes induced by the anæmia. The well-established fact that optic neuritis may result from chlorosis is interesting as proof of the intensity of changes in nerve-structures which anæmia may excite. The cases of chlorotic paraplegia are very rare, and need further study.

In the *quantitative anæmia* that results from loss of blood, besides the symptoms just described, paraplegia sometimes comes on, under conditions which exclude the idea of hysteria. The source of the hæmorrhage has most frequently been the stomach, kidneys, and uterus. The paralysis is usually motor only, but Leyden has observed accompanying hyperæsthesia. It may come on a few hours or days after the loss of blood, or only at the end of one or two weeks. Most cases recover. The pathological process which causes the paralysis is not known. The loss of power is comparable to the loss of sight which occurs from the same cause, and in which there may be no visible morbid change, or inflammation may be found, sometimes succeeding the loss of sight, and therefore to be regarded as a result of the derangement of the nerve-elements, or of the influence to which this is due, rather than the cause of the amaurosis.

The treatment of the symptoms due to general anæmia is of course essentially the improvement of the blood-state. Symptoms which can be ascribed, with probability, to vaso-motor spasm may be prevented by drugs that cause a more uniform arterial tone, such as digitalis or belladonna, coupled with nux vomica or strychnia, to render the effect permanent.

Hyperæmia of the Spinal Cord.

The conditions of the return of venous blood from the cord probably shield it effectually from the mechanical congestion from which almost every other organ of the body suffers when the movement of blood through the thorax is hindered. The chief mechanical congestion to which the cord is liable is that which results from the influence of gravitation, and occurs when a person is lying on the back. The distension of the veins outside the dura mater, and also of those of the pia mater, which occurs when the heart has ceased to act, and the blood is free to obey the only mechanical force which then acts upon it, sufficiently proves the power of gravitation to congest the cord. But it is not probable that gravitation has anything like the same influence during life, counteracted, as it then is, by many other forces.

Some persons with imperfectly nourished cords (easily fatigued, liable to tingling, &c., in the legs) suffer from aching in the legs or spine when they lie on the back. Since this ceases when the posture is changed, it is probably due to the mechanical congestion of the cord or membranes. It is doubtful whether any other symptoms can be, with confidence, assigned to mechanical congestion.

Of active congestion our definite knowledge may be summed up in the statement that it occurs in the early stage of inflammation. We know nothing of it as an independent condition. Nevertheless many pages might be filled by the enumeration of the various symptoms of congestion of the spinal cord,—in which the unrestricted play of "scientific" fancy has elaborated a symptomatology for the separate congestion of every part of the spinal cord. Probably the only case in which we are warranted in suspecting a primary active congestion of the cord or membranes, is that in which symptoms identical with those of commencing myelitis, and following a cause of myelitis, pass away in the course of a few days. These cases will be mentioned in the account of inflammation of the cord.

An active congestion, which may be called secondary, seems to result from prolonged and violent action of the nerve-elements of the spinal cord. Thus local dilatation and distension of vessels, migration of leucocytes into the sheath and into the adjacent tissue, and even small extravasations, are found in some cases of hydrophobia, of tetanus, and of strychnine poisoning, and it is probable that a similar congestion attends all violent physiological activity, violent and continued muscular exertion, coitus, &c. Coitus several times repeated has been known to cause hæmorrhage within the cord which must be preceded by an intense congestion. Vascular dilatation, with an increased blood-supply, is the physiological attendant of functional activity in all organs, and doubtless also in the cord, but it is morbid only when excessive. It is doubtful whether it causes symptoms unless it goes on, as it probably may, to actual inflammation.

INFLAMMATION OF THE SPINAL CORD; MYELITIS.

Inflammation of the spinal cord is a common lesion in the acute, subacute, and chronic forms. It usually causes a considerable diminution of consistence, and hence the term "softening of the spinal cord" has been used as a synonym for it. But the term is not accurate, because inflammation does not always cause softening, and a diminution of consistence may occur without inflammation.

The spinal cord differs from most other structures in that its power of recovering functional capacity after damage by inflammation is comparatively small. The symptoms persist for a long time, and are not unfrequently permanent. Hence the distinction between acute and chronic inflammation is to be drawn, not from the whole course of the disease, but from the mode of onset. It is this which indicates the character of the morbid process. *Acute myelitis* is that form in which the symptoms indicating inflammation of the cord come on rapidly and reach a considerable height in a short time, in less than two weeks. When the onset occupies from two to six weeks the inflammation is regarded as *subacute.* When a still longer time elapses before the symptoms reach a considerable degree of intensity the myelitis is termed *chronic.* There is, of course, no sharp distinction between these classes. The subacute and acute forms may be conveniently described together. Other varieties are based on the distribution of the disease. The grey matter may be affected only or chiefly, *polio-myelitis* (from πολιος, grey), a form that has distinctive characters which make its separate description desirable. When the whole thickness of the cord is affected in a small vertical extent the myelitis is said to be *transverse.* This is one of the most common forms. When an extensive area of the cord is continuously inflamed the myelitis is said to be *diffuse;* when one small area is affected the inflammation is called *focal;* when there are many foci contiguous or distant the myelitis is said to be *disseminated.* Inflammation of the grey matter, around the central canal, extending into the intermediate grey substance and to a less extent into the cornua, has been termed *central myelitis.* A further important distinction is that between *parenchymatous* myelitis, an acute affection beginning in the nerve-elements, and the *interstitial* form, which begins either in all the elements or in the connecting tissue, and in which vascular disturbance takes a prominent part. The latter is the common form of myelitis, whether transverse or disseminated, but the former is of importance on account of the limited class of symptoms to which it gives rise and of its special causal relations. This distinction is already familiar in regard to multiple inflammation in the nerves.

ACUTE MYELITIS.

ETIOLOGY.—*Remote causes.*—Little is known of the influence of hereditary predisposition, but myelitis is sometimes met with in members of families in which other evidence of a neuropathic tendency can be traced. Males suffer more frequently than females. It may occur at all ages, but the form which occurs in young children is usually confined to the anterior cornua; other forms of myelitis are most common between ten and forty years.

Immediate causes.—Myelitis, acute and subacute, is readily produced by injuries to the cord of all kinds,—lacerations, bruises, and punctured wounds. It also seems to follow simple concussion, but in many of these cases there have been minute foci of structural damage. The myelitis produced by concussion is often, however, far more extensive than the primary lesion, for the latter may cause few symptoms, and a few days later a complete transverse myelitis may develop. Hæmorrhage into the cord, especially of traumatic origin, may have a similar effect; it may be followed after a short interval by extensive myelitis. In cases in which an interval of more than a few days elapses, pain in the spine may show that a morbid process is going on by which the myelitis is produced. Thus, in one case of acute myelitis, two weeks after a fall, there was continuous pain during the interval, corresponding to the seat of the later inflammation. Concussion also sets up the subacute form of myelitis, and this is especially common after railway accidents (see Traumatic Lesions).

Allied to injury as a cause is violent action of the muscles attached to the spinal column, which has sometimes appeared to be the sole agent in producing an acute inflammation in the adjacent part of the spinal cord. The disease has been known to follow a single violent contraction of the dorsal muscles, as, for instance, a successful but violent effort to prevent a fall. It is possible that there is an actual injury to the cord in these cases, but more probably inflammation is set up in the fibrous tissues of the vertebral column or nerve-roots and spreads to the cord. It is also possible that compression of the vessels may be concerned in the mechanism. In several cases the disease has resulted from attempts to lift heavy weights. Thus a man, two days after such an attempt, became rapidly paraplegic, and ultimately died; softening of the cord was found at the level of the fifth and sixth cervical vertebræ. Slow compression (*q. v.*) is constantly attended by inflammation in the compressed part; the myelitis thus set up may have an independent course, acute or subacute, and may not be proportioned to the pressure. In cancer of the vertebral bones, for instance, very slight pressure may cause an acute inflammation; there is probably sometimes an extension of inflammation to the cord from the external disease, but such extension is not necessary for the occurrence of the myelitis; the inner surface of the dura mater is often normal. Acute myelitis also results from internal meningitis, in which some degree of invasion of the cord is almost invariable.

Another exciting cause is exposure to cold, especially when the body is heated, lying on damp grass or on snow, bathing in cold water, &c. This cause has been supposed to act by suppression of the functions of the skin, since myelitis has been produced by varnishing the skin of animals,* but it is unlikely that this is the sole mechanism.

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* Feinberg, 'Virchow's Archiv,' Bd. lix. In Vulpian's laboratory similar experiments failed.

Exposure of the back to cold sometimes seems especially effective. Myelitis has been produced experimentally in rabbits by freezing the skin of the back with ether spray (Feinberg). It is probable that cold usually acts by the mechanism of a morbid blood-state, such as that which causes rheumatic fever, but which certainly varies greatly in its precise character and pathogenetic power. It is also certain that local exposure frequently causes inflammation in organs corresponding to the part of the surface that is especially chilled, although this influence may be chiefly to determine the incidence of the effect of the blood-state. In myelitis from cold, local exposure can seldom be traced, and the common mechanism is probably that of a blood-state. It is instructive to compare the analogous facts regarding the causation of multiple neuritis. Over-exertion is another powerful cause, and is especially effective when combined with insufficient food, and exposure to cold.

The sudden suppression of the menses and of other habitual discharges has been supposed to cause the disease. In most instances of the kind there has also been exposure to cold, to which the suppression and the myelitis may both have been due, and it is impossible to say how far the former has been concerned in the process. Thus, a girl sat on wet grass during menstruation, which was immediately arrested, and myelitis followed, a day or two later. It is conceivable that the sudden arrest of the discharge may lead to a morbid state of the blood, or modify that which a chill alone is capable of producing, and that the derangement of the uterine functions may have a direct influence on the cord and dispose it to suffer. It is noteworthy that, in the case referred to, the inflammation was in the part of the cord to which the pelvic organs are related, the lumbar region.

Functional over-action of the cord may cause inflammation in the part that is thus over-exerted. It has been already mentioned that long-continued stimulation of the sensory nerves may cause myelitis at the spot related to the nerves stimulated. As we have just seen, active function everywhere involves vascular dilatation, which, if excessive, amounts to active congestion, and, when prolonged, is attended by the escape of leucocytes that renders the condition one of inflammation. However excited, such a process has a tendency to independence of course, and to proceed beyond the degree corresponding to its cause. It is especially local functional over-action that has a tendency to cause myelitis. Thus the affection seems to be sometimes produced by sexual excesses. Occasionally, however, general over-action of the cord, as in severe, long-continued exertion, has been followed by the disease. In one case, it was due to lying to rest on damp ground, after an unusually long walk on a hot day.

Toxic blood-states constitute a well-marked class of causes, and their influence is rendered the more distinct by the analogous causaion of multiple neuritis. They may be acute conditions due to some rganised virus, perhaps allied to those that produce the acute specific iseases. Inflammation of the cord occasionally comes on in the ourse of, or after, typhus, typhoid, and especially variola and neasles, as well as acute rheumatism and severe puerperal diseases. he onset may be during the acute disease, at any period after it ias existed for about a third of its duration, or it may occur during onvalescence, or a month or more after the disease is over. Whether yphilis is a cause of acute myelitis is a question on which opinions iffer. Attacks of acute myelitis are not uncommon in syphilitic subjects, and it seems on the whole probable that there is a causal relation to syphilis, although it remains to be ascertained whether the mechanism is by disease of the vessels, such as is found in the pia mater in some cases,* or whether it is by the agency of a blood-state. The myelitis that occurs in acute specific diseases is probably due to the virus of the primary disease, or to a product of it, and the fact that the inflammation is commonly disseminated suggests that some specific organisms have a tendency to fix themselves in the spinal cord and set up inflammation. In diphtheritic paralysis the cells of the cord may present changes that amount to a parenchymatous myelitis. The fact that chronic changes in the cord may be a late effect of syphilis (as in tabes) makes it the more easy to conceive that acute processes should sometimes occur in an earlier stage of the disease.

In another class of diseases, a special influence of the virus appears to be associated with the results of functional over-action. This effect is especially seen in hydrophobia. In some cases of this disease, foci of a somewhat diffuse myelitis of slight degree are conspicuous on microscopical examination. Still more intense myelitic changes are sometimes met with in the chorea of the dog, although, in other instances, the cord is normal.

Chronic blood-states and constitutional conditions may also cause acute myelitis. Alcoholism is an occasional cause, and frequently produces the subacute form. The most potent agent of this class is gout. This is known to be a frequent cause of inflammation in various structures,—of the nerves among the rest; and cases of acute and subacute myelitis are met with, especially of the disseminated and relapsing forms, for which no other cause can be discovered. In one such case the patient had but lately recovered from a severe and prolonged attack of gouty inflammation of several intraocular structures. Like other chronic general causes, gout probably often acts as a predisponent when the malady is excited by some other cause.

Irritation and inflammation of peripheral organs, as the uterus, bladder, kidneys, intestine, &c., has been supposed to be an occasional cause, but this influence is exceedingly doubtful, except in the rare cases in which inflammation of a nerve ascends to the cord. In a considerable number of cases we are able to discover no cause to which myelitis can be ascribed. Many of these are probably due to a blood-state which may have no other consequences or may also cause simultaneous inflammation elsewhere.

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* Sudden paraplegia occurred in a case in which there was diffuse arteritis, supposed to be syphilitic (Schmaus, 'Arch. f. kl. Med.,' 1889).

PATHOLOGICAL ANATOMY.—In recent acute inflammation of the cord, the pia mater, at the affected part, is usually red and vascular, and the cord itself may be slightly swollen. Its tissue is reddened and often marbled from distended vessels, and the distinction between white and grey matter is indistinct and lost.

The diminution of consistence which occurs in acute inflammation may be so great that the affected part is diffluent, like cream, and flows out when the pia mater is divided. It does not follow that the consistence is lessened to this degree during life; post-mortem softening occurs more readily at an inflamed part. When the vascular disturbance and extravasation are considerable, the condition is termed "red softening;" so much blood may be effused into the tissue that the part looks as if there had been an actual hæmorrhage, and in some cases an extensive extravasation does actually exist; this form is termed "hæmorrhagic myelitis." If the inflammation has existed for some time, the effused blood is altered in tint, and the colour of the softened part may be rather chocolate than red. After a longer time the changes in the blood-pigment cause the colour to be lighter, "yellow softening." In many cases the breaking up of the nerve-elements (partly, as just stated, post mortem) is out of all proportion to the extravasation of blood, so that the softening is "white." It is said that red and yellow softening may ultimately become white from the removal of the blood-pigment, but the evidence of this is inadequate. The period at which these changes of colour are found varies according to the amount of extravasation at the onset. At a later period the yellow or white opaque aspect is changed to a grey translucent appearance, from the absorption of the fatty products of degeneration, and an increase in the connective tissue. If the myelitis is very limited in extent, cavities may be formed within the cord.

The microscopical appearances in myelitis vary much according to the duration and form of the inflammation. In the fresh state the most conspicuous objects are the products of degeneration of the fibres, granules and masses of myelin, and granule corpuscles, with, in older cases, the peculiar bodies termed "corpora amylacea" (Fig. 93, *d*). With these are numerous red blood-discs, leucocyte-like corpuscles, and, after the first week, distinct cells, round, oval, spindle-shaped, or angular, with nuclei which resemble leucocytes in size, and are readily mistaken for them. Fragments of axis-cylinders may also be seen, granular in aspect, and with irregular swellings (Fig. 93, *a*).

The softened and even diffluent state of the tissue prevents us from learning much, by microscopical examination, of the condition of the parts in which the inflammation is intense and diffuse, because the breaking up of the tissue and separation of the fragments renders it impossible to obtain sections of the hardened organ. It is only where the inflammation is less intense, or less uniformly diffused, that a thorough examination can be made. The products of degeneration are best seen in the fresh state or in sections mounted in glycerine (Fig 93). The fragments and globules of myelin in part occupy the position of the nerve-fibres from which they have come, in part are aggregated into masses.

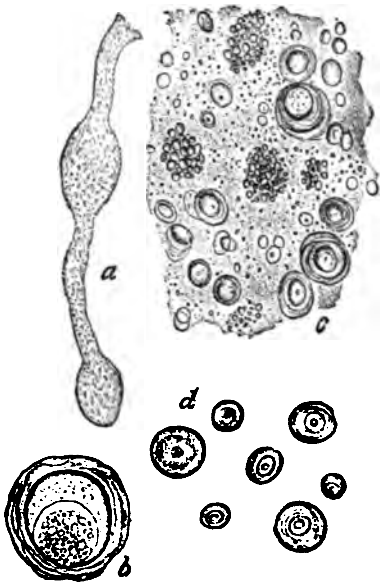

FIG. 93.—Myelitis. *a*, an irregularly swollen axis-cylinder. *b*, section of nerve-fibre with swollen axis-cylinder. *c*, section of white substance, with granule-masses and atrophied nerve-fibres. *d*, corpora amylacea. (From Leyden.)

In sections rendered transparent in the ordinary way, the fatty products of the destruction of the nerve-elements are invisible, and the most conspicuous alterations are the dilatation of vessels, which is often very great, and the accumulation of leucocytes and other cell-elements in the tissue and especially about the vessels. In the white substance the larger vessels are conspicuous by their enlargement, and in the grey substance the dilated arteries and capillaries may occupy a considerable part of the tissue (Figs. 94, 95, A). The nuclei of the capillaries are large and numerous. The walls of the smaller arteries are encrusted with leucocytes in the early stage, and, later on, in both white and grey substance, are much thickened by cells, the nuclei of which are often elongated and more or less concentric to the lumen of the vessel (Fig. 98, D). Outside this thickened wall the perivascular sheath is enormously distended, at first by leucocytes, among which, afterwards, other cells are mingled, round, fusiform, angular, with leucocyte-like nuclei (Fig. 98). In transverse section this distended sheath may look, at first sight, like an enormously thickened wall. Red blood-corpuscles distend the vessels and are seen also in the adjacent tissue, sometimes uniformly scattered through it (Fig. 98, F), sometimes aggregated in small extravasations due to the rupture of minute vessels. The grey substance is densely set with round corpuscles, staining deeply, many of which are the nuclei of small fusiform or angular cells (Figs. 95, A; 97. B) while the intervening substance is much more granular than in health. The nerve-cells are much swollen and granular, often contain distinct globules, strongly refracting, and probably fatty. The cell-processes appear shrivelled or lost (Fig. 95, A). In slight cases the margins of the cells may be less sharply defined than in health, and the bodies then may contain large vacuoles, sometimes wholly within the cells, some-

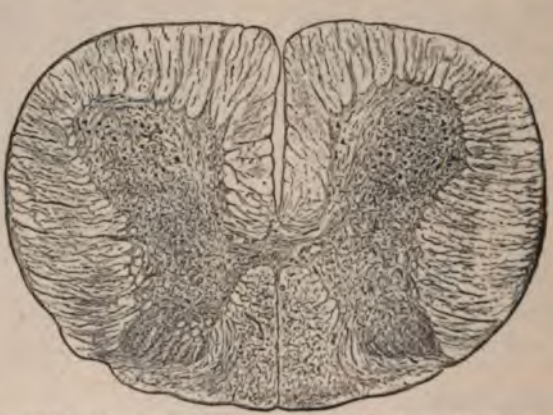

FIG. 94.—Subacute myelitis, lumbar region; both white and grey substance uniformly affected. In the latter the numerous distended vessels appear as lines; here and there a larger one is visible.

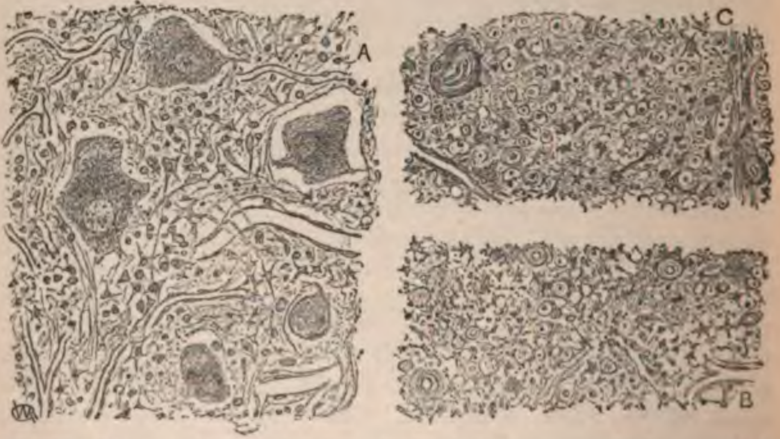

FIG. 95.—Portions of the section shown in the last figure, more highly magnified A, from the left ant. cornu; distended capillaries with numerous nuclei; larger vessels with slight distension of perivascular sheaths; numerous round and stellate cells; ganglion-cells, swollen, with shrivelled processes. C, from front of post. column; scattered nerve-fibres separated by amorphous material in which small cells can be seen, round and irregular, some distinct, others indistinct, vessels with thickened walls. B, from near the posterior surface, shows similar changes but with more open spaces from which nerve-fibres have perished. (From sections prepared by Dr. Money.)

times partly within and partly outside them in an adjacent albuminous-looking substance.* In some cases, however, especially those of disseminated interstitial myelitis of moderate degree, the nerve-cells appear little changed in hardened and cleared specimens, although a granular condition can usually be seen in the recent state.

In the white substance, cleared sections show a great increase and alteration in the interstitial tissue. The change may be uniformly distributed, or greatest in the neighbourhood of the vessels (Fig. 98, c). The increase is due in part to an amorphous material, containing nuclei here and there, some of which belong to cells such as have been described in the grey substance; and these may constitute extensive tracts between the spaces where nerve-fibres once existed (Fig. 98). In older cases the tissue may have a fibrous aspect in places. The large cells, with many processes called "spider cells," or "cells of Deiters," are often conspicuous objects (Fig. 96). The processes extend between the nerve-fibres. We have seen (p. 165) that their existence in the normal cord has been doubted, but in the inflamed cord, in which they are enlarged, their presence is distinct.

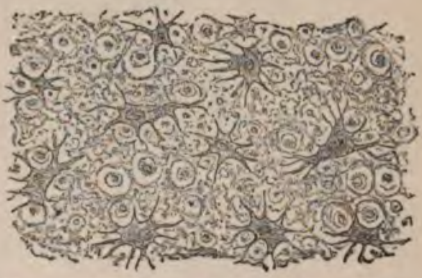

FIG. 96.—Subacute disseminated myelitis; part of the posterior column. Nerve-fibres separated by granular material and products of degeneration, and by numerous large spider cells. The myelitis in this case was attended by optic neuritis. (From a section lent by Dr. Dreschfeld.)

The changes in the nerve-fibres vary much. They always suffer destructive changes, but the products of their degeneration, above described, are scarcely to be seen in cleared specimens. The slightest change in the myelin appears to consist in an alteration in its chemical nature, in consequence of which it stains more readily than in health. Fibres are thus changed adjacent to any increase in the interstitial tissue, and they contrast with the unstained healthy fibres in the vicinity (Fig. 97, *c*). With or without this change in the myelin, there is an irregular swelling of the axis-cylinders, such as is seen on examination in the fresh state. If the fibres are divided transversely at the places where the axis-cylinder is swollen, this appears much larger than normal (Fig. 93, *b*). Examples of this will be found in most of the figures. These intense degenerative changes cause the breaking up of the myelin, and may involve the destruction of the fibre, the space occupied by it being left empty.

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* The significance of this vacuolation has been much discussed. It is probable that the vacuoles form after death, perhaps during the process of hardening, but that they do not form unless the cells have been altered by disease. They have thus some pathological significance.

In other cases, however, the axis-cylinder persists and remains surrounded by a narrow zone of myelin. Often a large number of such shrunken fibres may be found in a part which at first sight appeared devoid of fibres. This shrinking of the fibres occurs not only as the result of pressure on them by the connective-tissue elements, but also where there is little interstitial change and no compression.

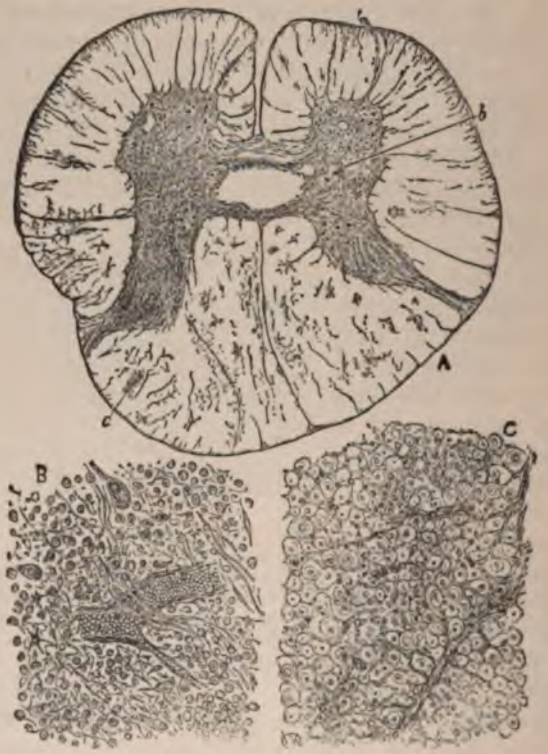

FIG. 97.—Acute transverse myelitis, fatal in three weeks. Carmine preparations. A, section through most diseased part, at first lumbar segment. Foci of inflammation scattered through the whole area of this cord, most abundant in the posterior columns (the apparent enlargement of which is probably due to the direction of the section). Dilatation of the central canal. B, from the anterior cornu at A, *b*, a distended vessel; the tissue crammed with lymphoid and other cells, among which run normal nerve-fibres. C, from the posterior column, A, *c*. The areas staining deeply are seen to consist of thickened trabeculæ, lymphoid cells, amorphous tissue, and also of nerve-fibres, the white substance of which stains with carmine while that of the neighbouring healthy fibres does not.[*]

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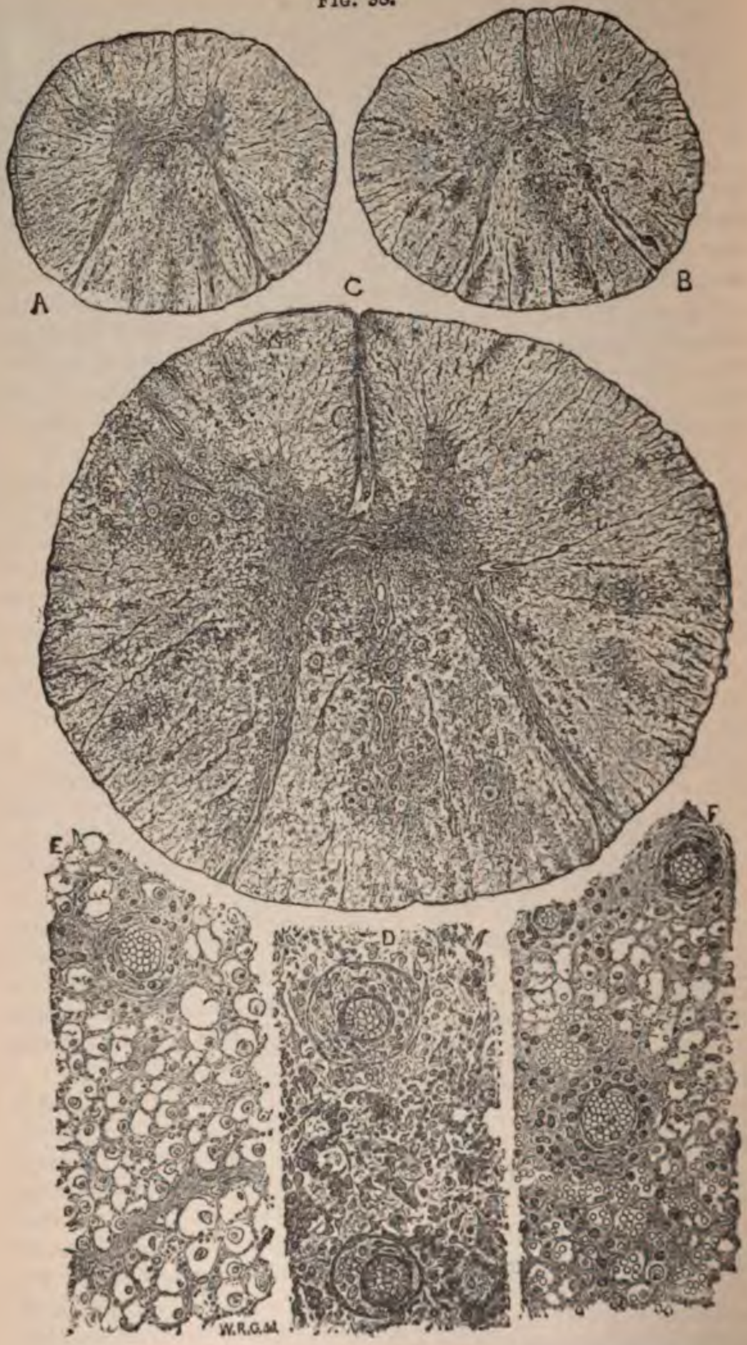
[*] I am indebted to Dr. Dreschfeld for the section from which these drawings are made. The patient was a girl aged twenty-three; paralysis, motor and sensory, was complete at the end of two days; the application by her mother of hot bottles to the feet caused such extensive sloughing ulcers that amputation of one leg was deemed necessary three weeks after the onset, and the patient died the next day.

The relative amount of change in the nerve-elements and in the interstitial tissue varies very much. In some cases the interstitial changes are evidently primary, and the fibres at first present little change in slight cases, but afterwards are altered secondarily, being narrowed, and even destroyed, as they are surrounded and enclosed by the products of the inflammation. In other places, or even other parts of the same section, there is but little increase in the neuroglia, although the fibres have suffered extensively; empty spaces are limited by septa but little thicker than normal, although with more nuclei, and often with numerous granules, the nature of which is uncertain. Occasionally there is a more diffuse change in the white substance; the neuroglial tissue is increased by small cells and amorphous material in such a manner that all appearance of septa and spaces is lost; a few nerve-fibres are included in it, and the spider cells are very conspicuous objects, their processes apparently representing the septa that exist in the normal condition (Fig. 96).

The distribution of these changes is very variable. The whole thickness of the cord is uniformly affected in pressure-myelitis (see "Compression of the Spinal Cord"). In transverse myelitis the changes may be so intense in all the structures at the affected level that the cord is diffluent, and, as already stated, only separated products of degeneration and inflammation, and blood-corpuscles are to be seen. In slighter cases the alteration is seldom distributed uniformly, and in either a single section, or a series of sections taken near together, each part may present variations in the damage, which may, however, be such that no part escapes in all (see Fig. 98). In meningo-myelitis the periphery of the cord is always most affected, and in old cases the thickened pia mater may send tracts of dense tissue into the interior of the cord, from which branching processes of tissue may pass on all sides. The condition is similar to that met with in chronic myelitis. A similar extension is often conspicuous in cases in which there is no special inflammation of the membranes, and the interstitial changes often extend from the septa that normally pass into the cord and from the walls of the vessels, the course of which may thus be marked out when the vessels cannot be seen. Hence the white columns may be broken up by lines of tissue passing from the surface of the cord towards the grey substance.

In disseminated myelitis, the foci of inflammation may exist through a wide extent of the cord and appear on the surface as reddish-grey areas, closely resembling those of disseminated sclerosis, but less sharply outlined. Under the microscope, in the early stage, smaller foci stud the grey and white substance irregularly, sometimes extending into larger tracts. They may be most abundant in the grey or the white substance. At first the vascular changes are the more conspicuous, but afterwards the evidence of destruction of nerve-elements and the inflammatory products, exudative and cellular, described above, characterise these foci, while ultimately a condition

Fig. 98.



of irregular focal sclerosis remains. In central myelitis there is a great increase in the nuclei about the central canal, with vascular changes in the commissure adjacent, but there may also be evidence of slight widely-diffused myelitis. It is necessary to remember that the nuclei around the central tissue vary much in number, and that it is easy to mistake a normal for a diseased condition.

Little is known of the morbid appearances in the parenchymatous forms, except that in a variety of anterior polio-myelitis, and in diphtheritic and other toxic paralyses, the nerve-cells present granular changes, and the interstitial grey substance may be but slightly affected.

The nerve-roots coming from a much inflamed part are usually damaged, and may present histological changes similar to those within the cord, distension of vessels, increase of nuclei and of connective tissue about the vessels, breaking up of the myelin, swelling of the axis-cylinders. Degenerative and neuritic changes may descend the motor nerves, although rarely to the same degree as in polio-myelitis, except in the cases in which there is extensive inflammation in the lumbar or cervical enlargement.

From the foci of inflammation, ascending and descending degenerations pass along the tracts of long fibres that have been already described, downwards in the pyramidal tracts, upwards in the posterior median columns, the direct cerebellar tracts, and the antero-lateral ascending tract. In cases of transverse myelitis, moreover, changes that are definitely inflammatory in nature may be traced in certain tracts for a short distance above or below the chief lesion; they may accompany the secondary degeneration, or may be found extending along a tract that degenerates in the opposite direction. Thus distinct inflammation may pass, for a short distance, up the pyramidal tracts, so that, an inch or two above the upper limit of the general inflammatory changes, an ascending inflammation of these tracts may be combined with an ascending degeneration of the tracts that always present secondary ascending degeneration.

If the patient survives, the new interstitial tissue slowly undergoes

FIG. 98.—Acute transverse myelitis; from a case fatal in a month. A, B, C, neighbouring sections from the inflamed part, in the mid-dorsal region, to show the varying distribution of the inflammation. (Stained with aniline blue black.) Distended vessels are all surrounded by thick zones of clear tissue, in places divided obliquely, while outside this, ramifying tracts of darker tissue extend into the white substance. In D, from the front of the posterior column, the changes are shown, more highly magnified. The zone around the vessels is seen to consist of the perivascular sheath distended by cellular elements, round, oval, fusiform, angular. The white substance is crammed with similar cells, and hardly any trace of nerve-fibres or of normal structure can be perceived. In E, from a less affected part of the lateral column, stained with carmine, the alveoli remain, although the tissue between them is thickened, and in many places studded with refracting granules and larger nuclei. In many spaces nerve-fibres are seen much smaller than normal in consequence of the wasting of the white substance. F represents a similar condition, with fewer nerve-fibres, and extensive infiltration of the alveoli with blood-corpuscles. (From sections prepared by Dr. Money. For the specimen I am indebted to Dr. Hadden.)

changes which give it a fibroid aspect, although for some time cell-forms may preponderate in it. We know very little of the nature of the process that occurs in the cases which slowly recover, and subserves restoration of function. The fact that a long period of total palsy may be succeeded by the slow return of considerable power, shows that even greatly damaged fibres may regain functional capacity. It is easy to conceive that the fibres which are only so far changed, that the axis-cylinders are swollen and the myelin stains readily, may speedily recover. We can also understand that the fibres in which a very narrow layer of myelin remains around the axis-cylinder, may also regain the power of conducting. We must presume that in these fibres there is no interruption of the axis-cylinders, but there are cases in which we can scarcely believe that the axis-cylinders retain their continuity, although conducting capacity is ultimately restored. An absolute motor palsy of twelve months' duration must depend on changes that involve an absolute interruption of the fibres, and yet in such a case some return of power may occur. Is this effected by a growth of new fibres such as occurs in nerves? In the lower animals such a growth of fibres has been proved to occur; in man it has not yet been demonstrated, but it is difficult to conceive any other explanation of the clinical facts. The very interesting appearance presented by the section of a cord shown in Figs. 99, B, and 100 suggests strongly a process of regeneration. The cord was crushed in the mid-dorsal region by a fracture of the spine, and motor and sensory paralysis remained absolute, up to the level of the lesion, till the patient's death, six months later. At the seventh cervical (Fig. 99, A) the cord presents only the usual ascending degenerations, but at the eighth cervical (Fig. 99, B) opaque tracts extend through the grey and white matter, and adjacent to these there is everywhere an extensive destruction of nerve-fibres. In these tracts within the lateral columns a large number of very minute nerve-fibres can be seen, each consisting of a fine axis-cylinder surrounded by myelin (Fig. 100, c). The fibres are smaller than any met with in the normal cord. The appearance is as if there had been an irregular ascending myelitis, which had extended up the cord as high as between the seventh and eighth cervical segments, and from the lower extremity of the normal fibres there had occurred a growth of new fibres such as effects the regeneration of nerves.* We seem to have here an actual process of renewal of fibres that had been destroyed by such inflammation as has caused the empty spaces in the vicinity. If this is a correct interpretation of the appearances, they constitute proof that, even in the human cord, it is possible to have a new formation of destroyed fibres. That pyramidal fibres may be renewed after they have been degenerated in consequence of pressure, there is abundant clinical evidence, since complete paralysis with

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* For these sections I am indebted to Dr. F. G. Penrose. Unfortunately I have been unable to obtain any other sections of this part of the cord.

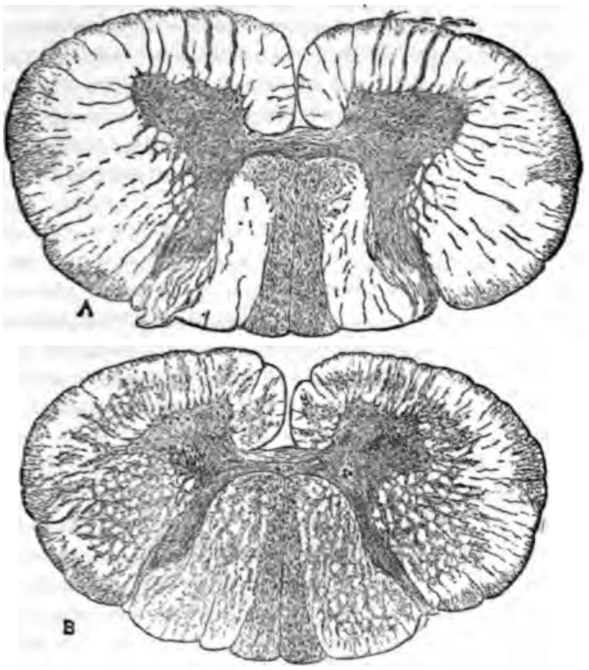

FIG. 99.—Ascending myelitis from fracture of spine completely destroying the cord in the mid-dorsal region. A, from the seventh cervical, presents only ascending degenerations in post. med. col., direct cerebel. areas, and ant.-lat. ascending tract. B, from the eighth cervical, presents also numerous areas of finely granular aspect under a low magnifying power, the finer structure of which is shown in Fig. 100.

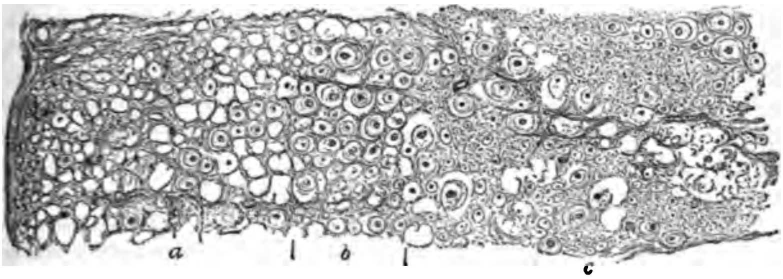

FIG. 100.—From the outer part of the front of the lateral column in B, Fig. 99. *a* is the antero-lateral ascending tract, the fibres completely degenerated, except a few, probably of other nature and belonging to *b*, a narrow zone, nearly healthy, of the antero-lateral ground fibres. *c* represents the outer part of one of the finely granular tracts. Under the higher magnifying power these tracts are seen to consist of very minute nerve-fibres with a granular material between them in which few cell-forms can be distinguished. Adjacent to these tracts there has apparently been extensive destruction of the fibres, large spaces being occupied by products of degeneration and appearing empty in the cleared section.

an intense spastic state of the limbs may be recovered from perfectly, even though it has lasted for many months. Such an event is common in cases of pressure-myelitis (*q. v.*), and a remarkable instance will be mentioned in the account of tumours of the cord. But in such cases, although it is not likely that the axis-cylinders of the fibres have preserved their integrity, the neuroglial space, which represents the nerve-sheath, has probably remained intact, and the re-growth of a fibre within it is a far simpler thing than a new formation of fibres in an area in which all the morphological framework has perished.

SYMPTOMS.—The symptoms of myelitis vary much according to its degree and form. The most common variety, which may be regarded as the typical form, is acute transverse myelitis, and to this especially the following description applies. The conspicuous symptoms are those which depend on the interference with the functions of the cord, and these, in most cases, are also the first indications of the disease. They are sometimes preceded, and more often accompanied, by general symptoms, such as attend inflammation of other internal organs—malaise, shivering, headache, depression, loss of appetite, pyrexia,—symptoms which may attend any form of acute myelitis, or may be altogether absent. When due to cold and commencing some days after exposure, general rheumatic pains may occur in the interval.

The spinal symptoms, if not prominent at first, quickly become so. The most obtrusive is the motor weakness, but this may be preceded, for a few hours or days, by sensory symptoms in the limbs—"numbness," tingling, or burning sensations. Occasionally there are rheumatoid pains at the onset, sometimes referred chiefly to the joints. Pain may be felt in the back, as well as in the limbs, but the vertebral pain is seldom prominent, and soon ceases. Occasionally spasmodic twitchings in the limbs or painful cramps attend the onset. In rare cases there is a general convulsion. This is most frequent in children, and then may be sometimes the expression of the general disturbance of the system. But convulsion occasionally attends the onset of acute myelitis in adults, even when there is no cerebral complication. Three instances of this have come under my own observation.

The motor paralysis usually comes on rapidly, and reaches a considerable degree in the course of a few hours. A patient after such sensations as have been described, or without any warning, finds that his legs feel heavy; after walking for a few hours he is obliged to sit down and rest. When he tries to walk again his legs feel "as if made of lead." He lies down for an hour, and then finds that he cannot stand, and in a few hours more is unable to raise his legs, although perhaps he can still move the feet or toes; next day, even this power may be lost. Occasionally the mode of onset is still more rapid, and occupies only a few minutes. The legs are found suddenly to be heavy and tingling, the sufferer sits down on a chair for a quarter of an hour, and then finds that he cannot stand. Such rapid onset resembles that of spinal hæmorrhage, and it is probable that in most of these cases there is hæmorrhage in addition to inflammation—

"hæmorrhagic myelitis." Such cases are, however, sufficiently rare not to interfere with the diagnostic rule that a sudden onset means a vascular lesion,* and that the characteristic onset of myelitis is rapid but not sudden. Now and then the onset is in the night, during sleep; a patient goes to bed well, and wakes up in the morning with complete paraplegia. On the other hand, the onset of the paralysis may occupy several days, or even a week; when longer than this, from ten to forty days, the myelitis is to be regarded as subacute. Occasionally the onset is by a series of sudden attacks of weakness at intervals of a few hours or days, or even weeks. In the latter case there may be some recovery from one attack before the next comes on. Frequently, there is gradual partial loss of power for a few days, and then complete paralysis comes on rapidly; the first symptoms may be slight, and their nature only recognised when the disease has developed.

When the paralysis has reached its height, and has ceased to increase, it is usually complete; sometimes it is incomplete, the limbs can be moved, but with little power, or the patient may be able to contract certain muscles but not to move the parts to which they are attached, perhaps only to move the toes. The distribution of the paralysis depends on the position and extent of the disease. In the majority of cases, the legs and lower part of the trunk only are affected, because the dorsal region of the cord is the most frequent seat of myelitis. If it is incomplete in the legs, the flexor muscles usually suffer more than the extensors, although this may not be apparent as the patient lies in bed, because the leverage at the knee renders necessary considerable power to extend the joint against gravitation. When the disease is in the cervical region the arms as well as the legs are involved, and the arms are sometimes paralysed before the legs. If the lesion is extensive in the cervical region, the damage to the grey matter and root-fibres causes atrophic palsy in the arms, while the legs are the seat of simple and ultimately spastic paralysis. In such cases, the intercostals are paralysed and breathing is carried on by the diaphragm. Myelitis above this level, if complete, abolishes all respiratory power, and necessarily causes death with great rapidity; but occasionally, in partial inflammation, the diaphragm may escape, or the intercostals retain some power and life be preserved. In such rare cases the arms as well as the legs may be the seat of simple palsy, and the neck muscles may become atrophied. This was seen in one patient, in whom the sterno-mastoids and the upper part of each trapezius were wasted, while the arms as well as the legs presented an increase of myotatic irritability. In such cases disseminated inflammation may spread into the medulla, the pharynx and the tongue may be affected, vomiting may occur, and the heart's action be interfered with. The range of motor symptoms, when the disease is at different heights, is indicated in the table of functions (p. 210).

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* Indeed, it is possible that these cases constitute no exception to the rule, the myelitis being set up by vascular obstruction.

Sensation is frequently impaired as well as motion. At the onset, indeed, it is involved in some degree in all cases of general transverse myelitis. In severe cases it is absolutely lost up to the level of the lesion. The upper limit of loss is often oblique, being higher in front than behind. In slighter cases the loss may be partial, and only certain forms of sensation may be impaired, either in consequence of the position of the disease, when touch or pain may be lost alone, or of its slight degree, when touch is generally impaired and painful sensibility preserved. Occasionally there is general hyperæsthesia in the limbs at the onset. At the level of the lesion, there is usually a zone of hyperæsthesia, corresponding to the distribution of the nerves that pass through the upper part of the affected region, *i. e.* through the lowest, merely irritated, portion of the cord above the disease. The hyperæsthesia may readily be detected by passing a hot sponge down the spine; the sense of warmth changes, at the hyperæsthetic zone, into one of pain. Corresponding to this zone of hyperæsthesia there is often a sense of painful constriction, a "girdle pain," which may persist for a long time, even after the loss of sensibility below the lesion has passed away. Occasionally it only comes on some time after the onset, probably from cicatricial compression and irritation of the root-fibres. Its position varies with that of the disease. It is most commonly felt between the umbilicus and ensiform cartilage, sometimes around the lower part of the abdomen, about the anus, or in the legs. It is of much practical importance because it is evidence of the existence of organic disease, and also of the upper limit of the lesion. It is sometimes a very early symptom. Any initial pain in the limbs usually ceases when sensation becomes abolished. When pain persists in the legs, it is often less when these are flexed.

The state of reflex action varies, and depends on the position of the disease, in accordance with the laws already stated (p. 220). An acute lesion in any part of the cord may cause an initial, inhibitory loss of reflex action in the parts below, but if the lesion is above the lumbar enlargement, reflex action returns in the course of a few hours. Frequently there is no initial depression. Subsequently the reflex action becomes excessive, that from the skin rapidly, that from the muscles more slowly. Ultimately each attains a high degree of exaltation. If the disease involves the lumbar enlargement all forms of reflex action are lost.[*] When in the cervical region, it may be lost in the arms, excessive in the legs. In dorsal myelitis the trunk-reflexes are often impaired, and may give important information as to the seat of the disease. The abdominal reflexes should be examined at various levels, and the greater impairment of these on one side than on the other may reveal clearly the unequal distribution of the lesion and its vertical extent.

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[*] See, however, the facts stated on p. 220. and 552.

The muscles of the legs are sometimes at first flabby and toneless during the stage of initial depression of reflex action, doubtless from the same influence. This condition soon passes off if the lesion is above the lumbar enlargement, and as reflex action returns, the muscles regain their tone. If, however, the myelitis involves the lumbar enlargement in considerable degree, the muscles of the legs remain flaccid, and lose all influence on the posture of the limbs. The feet fall into an extended position, so that the instep is in a line with the tibia (Fig. 101). The muscles also waste rapidly, and often present the reaction of degeneration. The wasting in these cases is often extreme. In many cases of myelitis above the lumbar enlargement there is a slighter and slower wasting of the muscles, without loss of reflex action and generally with excess of the muscle-reflexes; the ultimate amount of wasting may be considerable, but it is never so great as when the lumbar grey matter is diseased. There is never the reaction of degeneration, but only a slight change in irritability, similar to each current; first an increase, which, after some weeks or months, gives place to a slight diminution.

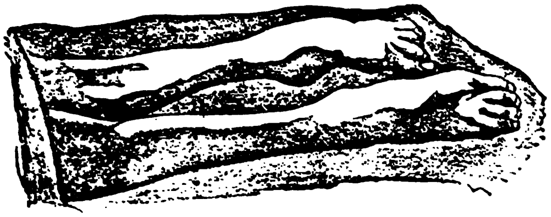

FIG. 101 —Posture of feet in myelitis of lumbar enlargement with rapid wasting of muscles.

The sphincters are usually affected from the first, except in very slight cases, and they often afford the earliest indication of the commencing lesion. There is usually first retention of urine, which not rarely exists for several hours, or even for a day or two, before other symptoms are added to it. There is afterwards incontinence, overflow or reflex (see p. 225), when the disease is above the lumbar region; if the lumbar centre is diseased there is persistent incontinence from the first without retention. In the former case there is involuntary action, in the latter inaction, of the sphincter ani, and in both cases incontinence of fæces. This may be inconspicuous in the early period, on account of constipation, due to intestinal paralysis. In cases of partial myelitis, as of the anterior cornua, the sphincters are often unaffected. The urine is frequently alkaline in reaction, and the change sometimes comes on so rapidly that it is evidently the result of an alteration in the secretion. In all cases of retention, decomposition in the bladder increases the alkalinity. Cystitis often follows, so that the urine contains pus.

The temperature of the paralysed limbs is usually at first raised one or two degrees above that of the mouth, but it afterwards falls, and remains a degree or so lower than that elsewhere. The skin is often dry, sometimes covered with sweat. Its nutrition often suffers, and bedsores result. In some cases they are due to long-continued pressure and neglect, but they may occur early and intensely when the disease affects the lumbar enlargement or irritation extends down to it from above. The least pressure on the skin may then cause a bulla containing sanious liquid, and sloughing occurs with readiness, especially over the sacrum. Now and then there is effusion into the knee-joints. Occasionally the tendency to sloughing of the skin is so intense that the lesions appear to be spontaneous.

Such trophic disturbances sometimes occur in the cellular tissue of the lower part of the body, especially in the neighbourhood of the bladder and rectum. A remarkable example of such disturbance was presented by the case from which Figs. 94 and 95 are taken. Symptoms of subacute myelitis were followed by those of cellulitis in the lower part of the abdomen, on account of which the patient was admitted into University College Hospital under the care of my colleague, Mr. Marcus Beck. Subcutaneous emphysema developed about the groins, and suppuration occurred near the rectum. In consequence of this inflammation the patient died, the cord symptoms having slowly increased; after death no lesion of the intestine or local cause of the cellulitis could be discovered. It is probable that the derangement of the trophic influence of the cord facilitates the occurrence of cystitis from retention of urine, and intensifies its effect on the kidneys. Ulceration of the bladder, which sometimes occurs with remarkable rapidity, may be thus produced, or a small abscess may form in the wall of the bladder, and thus an opening may be formed into the peritoneal cavity and fatal peritonitis may be induced. A vesico-vaginal fistula may be developed in the same way. Ulceration of the urethra, and consequent extravasation of urine, have been observed under similar conditions.* In another case the inflammation of the bladder was intense, and suppurative cellulitis occurred outside the organ and set up a fatal peritonitis; the kidneys were the seat of very acute suppurative inflammation.† These effects of cystitis may probably always be prevented.

When the disease is in the cervical region the pupil may be affected. In rare cases of myelitis, optic neuritis has been observed, without any intra-cranial complication to cause it.‡ It is probably not the result of the inflammation of the spinal cord, but is an associated and similar lesion, the result of the cause of the myelitis, which, in such cases, is probably a blood-state. It is noteworthy that most of the cases thus accompanied have been instances of disseminated myelitis, a form that suggests a cause acting widely on the nervous system. In one case* the optic neuritis reached its height some weeks before the occurrence of the first spinal symptoms, and in the cord were two separate and distant foci of inflammation; in another, the neuritis was found a fortnight before the onset of disseminated myelitis.†

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* S. West, 'St. Barth. Hosp. Rep.,' vol. x.

† Sharkey and Lawford, 'Trans. Ophth. Soc.,' 1883.

‡ Optic neuritis was present in the case of disseminated myelitis under the care of Dr. Dreschfeld from which Fig. 96 is taken, and also in that of Sharkey and Lawford just mentioned. See 'Medical Ophthalmoscopy,' 3rd ed., p. 189.

In cases of rapid development the degree attained by the symptoms at their onset may not be exceeded. In some cases a slower increase or extension ensues in the course of a few days. The myelitis, beginning below, may ascend the cord, and the arms and respiratory muscles may gradually become involved. The upward extension may be steadily progressive, or it may occur in successive separate attacks. In the latter case, it seldom extends above the dorsal region. Or inflammation, beginning in the dorsal region, may slowly descend into the lumbar enlargement. The extension may be by the grey matter, especially of the anterior cornua, or by the posterior region. In each case the extension downwards is indicated by loss of the reflex action present in the earlier stage: in the one there is rapid muscular wasting with loss of faradic irritability; in the other the muscles retain their nutrition and irritability, but sensation, if before present, becomes lost.

The constitutional symptoms, which have been mentioned as attending the onset, vary greatly in their severity and course. They are, as a rule, less in the simple transverse myelitis than in the disseminated form. In most cases they reach their height on the second or third day after the onset of severe symptoms. Thus in one case the temperature was normal on the first day of indisposition, when the patient complained only of slight headache and slight numbness of the legs. During the next three days these gradually became completely paralysed, and the temperature was successively 102°, 103°, and 104·5°, and then gradually fell to normal at the end of a week. In another case, in which the toxæmic state was part of that of measles, the temperature rose from 100° to 103·8° on the first day of the symptoms of myelitis, and on the next day to 104·2°, but fell (partly from antipyrin) on the third day, on which death occurred, to 100·2°.‡ In less acute cases, in which foci of inflammation develop progressively with spreading palsy, during one or two weeks, moderate irregular pyrexia, 99° to 101°, may go on as long as the disease is increasing, and may be renewed at any subsequent relapse. The temperature is also raised by any complications, and especially by cystitis. On the other hand, in simple cases, with a single focus of inflammation, the initial elevation may be trifling, and after the first day or two the temperature remains normal.

If the patient survives, the course of the symptoms varies according to the intensity of the lesion. The paralysis, motor and sensory, may remain complete. More frequently sensation is recovered after a few weeks or months, while motor power continues absent for a much longer period. The excessive reflex action may lead to the gradual development of spasm in the legs. This is especially related to the increase of the muscle-reflex action which follows the secondary descending degeneration of the pyramidal fibres. Ultimately the condition is that termed "spastic paraplegia," and described in detail in another section. The occurrence of this spasm is not incompatible with the recovery of some voluntary power. The muscles in these cases are often well nourished, and may even increase in size; the spasm constitutes a powerful stimulus to their growth. Spasm may, however, coexist with the slow moderate wasting described above (p. 223), but is rarely intense in these cases.

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* Sharkey and Lawford, op. cit.

† Achard and Guinon, 'Arch. de Méd. Exp.,' 1889.

‡ Barlow, 'Med.-Chir. Trans.,' 1887, p. 76.

In some cases, flexor spasm, after a time, takes the place of the extensor spasm, at first in transient attacks but at last permanently, and becomes fixed by contracture and shortening of the flexor muscles. It may be so great that the knees are against the abdomen and the heels against the nates. This substitution of flexor for extensor spasm occurs chiefly in cases in which the paralysis is absolute, and the organic disease of the cord so considerable and of such duration as to make recovery impossible. It is therefore a very grave sign.

Death may occur early in the disease from respiratory paralysis, or from the extension of disseminated myelitis into the medulla oblongata; subsequently it may be due to various effects of the trophic disturbance, acute or chronic, and to blood-states induced by bedsores or by kidney disease resulting from cystitis, &c., mechanisms that have been already mentioned in the account of the symptoms. Improvement, when it occurs, is usually slow, but continues for a long time, and recovery may be complete in cases of moderate severity. When there is much damage to the cord, however, recovery is often imperfect, and some weakness remains, accompanied either by spasm or by wasting. It is rare for there to be no improvement, but occasionally the palsy remains absolute, although life is prolonged for years. In some cases improvement occurs, slight or considerable, and is followed by a relapse, which leaves the patient worse than the first attack; and this may occur again and again, each relapse being the expression of a fresh extension of the inflammation in the cord. This relapsing course is met with especially in the disseminated form, and in the subjects of gout, either acquired or inherited. Other cases, again, present what may be termed a recurrent course, improvement, and perhaps a considerable degree of recovery, continuing for months, when a fresh attack occurs. On the other hand, in many cases, especially of simple transverse myelitis, when improvement is established it goes on, and there is no tendency to recurrence.

Varieties.—The different pathological forms of myelitis are attended by some differences in the symptoms. Those of *transverse* myelitis have been described as the most common and typical manifestations of the disease. *Focal* myelitis, in which there is a single spot of inflammation, not extending across the cord, may occur in various parts of the cord, but, on account of the slight degree of severity of the symptoms, little is known of them except when the focus of inflammation is of some size or is situated in one anterior cornu (as a variety of polio-myelitis), because such cases are rarely fatal. It is, however, probable that this variety is not uncommon. Cases are occasionally met with in which symptoms of very limited range come on acutely, and may reasonably be ascribed to such a lesion. They may be one-sided, and various in character, often being limited to a single limb.

A much more frequent focal form is *disseminated myelitis,* in which there are several foci of inflammation in the same or different parts of the cord. The onset of this form is often subacute, and constitutional symptoms are frequently absent. The most important special symptoms are those which indicate interference with the central functions of the cord in more than one locality, but the combinations produced are very variable. In many cases, the several foci of inflammation develop successively, not simultaneously, and then we have a corresponding succession of symptoms. Thus a myelitis in the cervical region, causing atrophy of some groups of muscles in one arm and paralysis of the corresponding leg, may be followed by paralysis of the other leg with such loss of reflex action in it as shows a separate focus of inflammation in the lumbar enlargement, and this again by the development of a girdle pain corresponding to the middle of the dorsal region, from a fresh area of inflammation in that situation. When numerous foci of myelitis occur in the dorsal region, as is not uncommon in syphilitic cases and after injury, the symptoms may closely resemble those of a transverse myelitis, because by one or another of these foci each of the elements of the cord is interrupted. The only distinction may be the extensive impairment of the trunk-reflexes, or the detection of loss of irritability in the corresponding muscles.

In the rare form of *diffuse central myelitis* there is usually rapid loss of power, of sensation, and of reflex action, considerable elevation of temperature, speedy trophic disturbance, and often death at the end of two or three days. Rarely, sensation and reflex action have been lowered and not lost. On the other hand, the loss of sensation is said to be sometimes absolute, when motor palsy is incomplete. The symptoms have begun in arms and legs simultaneously, or in either of these, and have accordingly spread upwards or downwards. Practically nothing is yet known of any slight non-fatal forms of this variety. *Hæmorrhagic myelitis* is scarcely a special form, since any acute inflammation of the cord may be attended by a sudden extrava-

[illegible] onset of severe [illegible] a commencing

[illegible] forms, and we have [illegible] analysis there is an [illegible] generally in the [illegible] lesion of the [illegible] nerves [illegible] affected [illegible] whether [illegible] that are of [illegible] extreme degree [illegible] normal [illegible] several instances [illegible] fibres [illegible] most organs in its great liability to primary inflammation, in which it presents a remarkable contrast to the brain. The liability is not equally distributed in the cord, but is least in the parts nearer the brain, less in the cervical than the lumbar enlargement, and less in these than in the dorsal region, so far as the most common form is concerned. The special liability of the dorsal region to transverse myelitis may be associated with the readiness with which this part undergoes post-mortem softening, but we are ignorant of the conditions by which this liability is determined. The disseminated form has less tendency to be localised in any special part. A question of considerable interest is whether acute inflammation of the cord always begins as such, or whether, in any case or form, it is set up by vascular obstruction, such as thrombosis in a minute vessel. It is conceivable that such an initial lesion may ultimately disappear in the intense inflammation it excites. Myelitis may be readily produced by the arrest of the blood-supply even for a short time, as numerous experimenters have shown, but their researches throw little light on the disease as it occurs in man; the effect is to produce first necrotic breaking up of the nerve-elements, and long after this the vascular lesions of ordinary interstitial myelitis. The processes are essentially different. But the experiments show that the lumbar enlargement is thus damaged with special readiness, and this suggests that the proclivity to disease of this part is due to its inherent susceptibility.

At present the questions of chief interest are the extent to which the disease is due to a morbid blood-state, the probable nature of such a cause, and the mechanism by which it acts. The chief facts that throw light on the subject have been mentioned already, the most important being the occurrence of myelitis in the acute specific diseases (in which we must ascribe it to the organised virus of the primary malady), and the evidence of an influence acting throughout the system, afforded by the wide extent of the disseminated form and its occasional association with disease elsewhere, such as optic neuritis. It is to this form that our knowledge chiefly relates. Of the pathology of simple transverse myelitis we have only the fact that it is often due to cold, which brings it into closer analogy with the simple inflammations of other organs.

The knowledge that has been gained of the influence of toxic blood-states on the peripheral nerves gives us the help of analogy in conceiving that myelitis may often have the same origin. But a difference exists in the fact that the multiple neuritis so produced is commonly parenchymatous, while the myelitis is conspicuously interstitial. Of the pathology of parenchymatous myelitis we know almost nothing. The question of causation will be again alluded to in considering the special inflammation of the grey matter.

The chief relation of the symptoms to the lesion has been considered in the general account of the symptoms of spinal cord disease, and those relating to the destructive and regenerating processes have been mentioned in the section on pathological anatomy. We may, however, further note that the vascular changes, which take so large a share in the process of interstitial inflammation, as seen, for instance, in the disseminated form, must extend the process. The escape of leucocytes into the sheaths tends to interfere with the lumen of the vessel and the flow through it, while their accumulation in the tissue must entail local destruction of the nerve-elements. If the cause of the myelitis is a persistent influence, such as the poison of a progressing specific disease, or the agent, whatever it be, that is effective in gout, it is easy to understand that the local influence of the lesion itself will co-operate with that of its cause, and indúce the progressive tendency that is a characteristic of this form.

DIAGNOSIS.—Myelitis is recognised by the rapid onset of symptoms indicating structural disease of the cord. Among these the failure of power is the most significant, although the subjective sensations that accompany it are often more obtrusive at the onset, and prevent mistake as to the meaning of the weakness. When they are absent the enfeeblement may be mistaken for mere prostration in cases in which some general illness coincides, and it is probable that slight myelitis sometimes occurs during an acute specific disease and altogether escapes detection. But among the early symptoms no one is more important, for its definite significance, than retention of urine; and its importance is increased by the fact that it may precede all symptoms in the legs and give a warning, the heed of which might sometimes enable lasting palsy to be prevented or life itself preserved. The diagnosis may be aided by the presence of such general symptoms as attend the occurrence of inflammation in other organs, but the absence of these is of little negative significance, while the presence of constitutional disturbance is as likely to mislead, as it is to suggest a local affection. The position of the myelitis must be inferred from the considerations already described. Its upper limit is indicated by the upper limit of the paralysis; that of sensation is most readily defined, and usually corresponds to that of motion. But the upper limit no more shows the extent of the disease below it, than the surface of water does its depth. The extent downwards must be gauged by the impairment of the functions of the cord as a central organ (reflex action and muscular excitability) in the parts paralysed, while the degree to which the various structures of the cord are damaged must be inferred from the character and degree of the symptoms in the affected parts. In ascertaining the state of the lower dorsal region the trunk and cremasteric reflexes are especially important.

Thus if called to a patient who has rapidly become paraplegic, the practitioner should first note the degree of motor and sensory paralysis of the legs (indicating whether the lesion is total or partial), and how high up the trunk the symptoms extend. He should then test the reflex action in the affected limbs and trunk, and ascertain the state of myotatic irritability in the limbs. These indicate the condition of the reflex arcs in the lower portion of the cord. Further information on this point is afforded by the state of muscular nutrition, and especially by the evidence of the state of nutrition of the nerve-fibres which is revealed by faradism. If reflex action is perfect and the muscles have preserved their tone, this examination is a matter rather of scientific interest than of practical importance. It is useless to apply this test until five or seven days after the onset, because four or five days, and often eight or ten, elapse before the degenerative changes occur in the nerve-fibres. The examination may then be made without any risk, provided a very gentle current is employed, just sufficient to cause a contraction in the corresponding muscles of a healthy limb, and the current need not be applied to each spot for more than a second. The isolated faradic shock may be employed with advantage on account of the absence of the strong sensory stimulation that is caused by the current (see "Atrophic Paralysis"). If no change is found, the examination may be repeated at the end of ten days from the onset. If no muscles, at the end of that time, present any considerable diminution of contractility, it shows that no considerable nutritive change is taking place in the nerves, and that the grey matter from which the nerves proceed is not inflamed. On the other hand, if certain muscles present a diminution of irritability, others being normal, there is a focal lesion in the corresponding grey matter. If all the muscles of both legs present such a failure there is inflammation of the lumbar grey matter, and if sensation is also lost, a total lumbar myelitis. So, if there is cervical myelitis with paralysis of all four limbs, the condition of irritability of the arm muscles shows whether the disease involves, or is above, the grey matter in the lower half of the cervical enlargement. If there is impairment of irritability, its extent in the two arms will afford an indication as to whether the myelitis is total, or whether it affects chiefly certain spots. The distinction from *simple polio-myelitis* depends on the fact that the symptoms are not purely motor. It is only when the myelitis is cervical or lumbar that the diagnosis is a matter of difficulty; in these cases the arms and legs are the seat of atrophic paralysis in ordinary myelitis, because the grey matter is involved. There is, indeed, polio-myelitis in such cases, but there is also more, and the impairment of sensation, together with the simple palsy of the legs when the disease is in the cervical region, shows the involvement of other parts than the anterior cornua,—which is the essential distinction. The most equivocal condition is that which exists when simple but severe polio-myelitis in the cervical region spreads to the white columns immediately contiguous, and causes weakness of the legs. But it never causes complete loss of power, even for a short time, and the legs soon recover, whereas in ordinary cervical myelitis the loss of power in the legs is often com-

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plete and prolonged, and is accompanied by impairment of sensation, not met with in polio-myelitis.

The distinction of the other varieties of myelitis rests on the special features already described. That of the *disseminated* form rests on the irregular distribution of the symptoms and the evidence of damage extending through a considerable vertical extent of the cord, but in some parts incomplete in degree. An inflammation which continues to extend after the first two or three days is certainly disseminated, and most subacute cases are of this variety, and so are those that are secondary to blood-states. The distinction is important, because this form is far more grave than any other, and more likely to cause death. It is doubtful whether *central* myelitis can be diagnosed during life; there is usually a slighter degree of myelitis in the other elements which causes symptoms resembling the ordinary form. There is, however, one exception to the rule that a spreading myelitis is disseminated; the diffuse transverse form that is secondary to some other morbid processes, either to compression from without, or to hæmorrhage within the cord, often undergoes subsequent extension. Acute myelitis in syphilitic subjects, even if due to syphilis, presents no distinguishing features, nor is its course influenced by antisyphilitic treatment. But the latter fact is true of all acute syphilitic inflammations—the tissue destroyed by the process cannot be restored by the removal of the cause. In rare cases, however, an acute parenchymatous myelitis in a syphilitic subject is suggested by the limited impairment of a certain function. In such cases, and in others in which the symptoms point to a toxic blood-state acting on the nerve-elements without destruction of tissue, the effect of treatment seems to confirm the diagnosis.

If transverse myelitis has been diagnosed, the question should always be asked, Is it primary, or secondary to some other process, of which the most common is compression of the cord? We have seen that myelitis may result, even in acute form, from external pressure, which may be that of a growth or of disease of the bone. The indications are, first, the fact that root-pains at the level of the disease preceded the myelitis for at least some weeks; and secondly, the direct evidence of a disease, as caries or cancer of the spine, to which the inflammation may be secondary. The spinal column should be carefully examined in all cases, not merely once, but again and again. When the myelitis is developed there may be no signs of bone disease, and yet these may appear in the course of a few weeks or months. Myelitis in a cancerous patient should always suggest a secondary growth in the spine. A woman, shortly after the removal of a cancer from the breast, became paraplegic; no evidence of bone disease could at first be found, but in a few weeks the vertebral column became distinctly enlarged from secondary cancer. This element in diagnosis has become of great importance from the certainty that some simple external growths can be removed.

The diagnosis from other lesions of the cord is chiefly by the mode of onset. In *hæmorrhage* the symptoms develop in a few minutes, and it is chiefly in the cases of hæmorrhagic myelitis that any diagnostic difficulty presents itself. The sudden paralysis is then commonly preceded by slight sensory symptoms, tingling, &c., and sometimes by fever. If these are absent in cases of actually sudden onset, primary myelitis is far less likely than hæmorrhage. The acute spinal pain that is common in hæmorrhage is absent in myelitis. If an onset indicating hæmorrhage is followed by a gradual extension of the symptoms during the next twelve or twenty-four hours, myelitis secondary to hæmorrhage may be inferred.

When acute myelitis ascends the cord, so that the legs, muscles of the trunk, and the arms are successively paralysed, its course resembles that of *acute ascending paralysis*, or "*Landry's paralysis*," in which no lesion of the cord is found after death. The most important distinction is that in ascending myelitis sensation is affected, and if the patient survives, there is a strong tendency to trophic disturbance in the skin, while many muscles waste and present loss of faradic irritability. In acute ascending paralysis, on the other hand, sensation is little impaired; some parts may be spared by the ascending palsy; bedsores do not form; and, if the case is not fatal, there is no change in the electric irritability of the muscles.

In *meningitis* the symptoms of irritation, severe pains, muscular rigidity, &c., are prominent, while they are absent in simple myelitis. But in many cases the two conditions co-exist, and which disease is predominant can only be decided by the order and degree of the development of the symptoms. In *meningeal hæmorrhage* there is severe pain in the back and acute irritation of the nerve-roots.

The distinction from *multiple neuritis* has been considered in the account of that disease. It chiefly arises in the cases of polio-myelitis, presently to be considered, but a difficulty may exist when a parenchymatous myelitis affects the structures on which co-ordination depends and produces the condition of myelitic ataxy. Such cases are distinguished from those of neuritic pseudo-tabes by the fact that some other structures are involved, so that symptoms are present that are only met with in diseases of the spinal cord. A very acute onset, so that the symptoms reach a high degree in a few hours, is also evidence of the myelitic origin. The case mentioned on p. 322 affords an illustration of these distinctions.

Myelitis is far from rare in patients of the age and sex in which hysteria especially prevails, and many cases are mistaken for *hysterical paraplegia*—sometimes because symptoms of hysteria concur, often merely because the patient is a girl and her legs well nourished. The converse error is very rare. The mistake occurs especially in cases of transverse dorsal myelitis, in which there is no wasting of the muscles of the legs. The gradual development of considerable excess of myotatic irritability is a symptom of great diagnostic value, and when this increases to characteristic extensor spasm there should be no room for doubt. If one leg is lifted from the bed, and the other moves with it owing to the rigid extensor spasm, organic disease is certain; hysterical contracture never fixes the legs to the pelvis so as to permit this effect. In hysterical paraplegia there may be retention of urine, but there is not incontinence. Among other conclusive symptoms of organic disease, a girdle pain, and incontinence of fæces, are of especial value. Trophic changes in the skin sometimes decide, even alone, the nature of the case.

PROGNOSIS.—The primary danger to life in myelitis depends upon the risk of respiratory palsy, and hence on the region of the cord in which the disease is situated. A disposition to spread is always of grave significance, and so also are indications of a serious blood-change, which is likely to increase the damage it has caused. On this account the prognosis is worse in disseminated than in simple transverse myelitis, and it is longer before confidence can be felt that arrest will endure. The risk is great whenever the cervical region is diseased, so that the intercostal muscles are paralysed, and in proportion to the nearness of the lesion to the origin of the phrenic nerve, on which life then depends. Hence a careful examination of the respiratory movements should be the first concern. Any indications that foci of inflammation have developed in the medulla oblongata are of extremely grave significance. Another danger is from acute trophic disturbances, and on this account the prognosis must be guarded whenever the lumbar enlargement is diseased, or is threatened by the extension of the inflammation. Hence bedsores, if forming early, within the first month, are an unfavorable indication; at a later period they have less influence on the prognosis. The occurrence of cystitis, and especially of any indication of secondary kidney disease, increases very much the gravity of the case.

The prospect of recovery of power chiefly arises when the onset is over, and the disease has become stationary. It depends (1) on the intensity of the disease, as shown especially by the loss of sensation as well as of motion, and (2) on its vertical extent as indicated by the impairment of the central functions of the cord; (3) on the early occurrence of symptoms of improvement. The longer loss of sensation continues, the less is likely to be the degree of ultimate recovery. (4) It is influenced to some extent by the cause of the myelitis, being better if this is due to a removable cause, such as pressure, than if spontaneous. (5) It is better when the disease is confined to the dorsal region than when rapid atrophy, &c., of the muscles show that the lumbar grey matter is involved. The longer motor palsy remains absolute, the less perfect will be the ultimate recovery. If some power returns within a fortnight, the amount that will be regained will probably be great; but even complete loss of power for six months does not preclude the ultimate return of the ability to walk, and I have even known this result after complete paralysis for a year from transverse myelitis. The development of increased myotatic irritability shows that improvement will not speedily occur, but even rigidity and the state of "spastic paraplegia" does not lessen materially the prospect of some recovery in a case in which the loss of power remains complete for a month or more. Indeed, the spasm often enables the patient to stand with a slighter degree of voluntary power than would suffice if the limbs were supple.

The fact of preceding syphilis must be allowed very little influence on the prognosis in a case of simple acute myelitis, but greatly improves the prognosis if there are indications that the inflammation is secondary to a more chronic process outside the cord.

TREATMENT.—If a case comes under observation at the earliest period, when only slight sensory disturbance and slight weakness of the legs indicate the commencing process, the question arises whether any treatment can avert the further development of the inflammation. If the symptoms are clearly due to exposure to cold, a hot bath, followed by free diaphoresis, should be employed, and followed by counter-irritation and the other measures now described. If considerable paralysis shows that the process of inflammation is fully developed, little can be expected from these measures, and it is better not to subject the patient to treatment that is incompatible with perfect rest. This, in all cases, is of paramount importance. Both functional excitation of the cord and movement of the spinal column should be avoided. The remarks regarding posture made in the account of the treatment of inflammation of the membranes apply also to that of the cord itself; it is most undesirable that the spine should be the lowest part of the body, and it is rather less difficult to keep the patient off the back in myelitis than in meningitis. A plank back-rest in the bed will be found a great assistance in securing comfortable rest on the side. If there is any reason to suspect hæmorrhage, or if there are indications of rapid extension of the inflammation, the prone position should be adopted, and even in myelitis it is well to adopt it at times as a change from the lateral posture.

The removal of blood from the skin of the back over the affected region, by leeches or wet cupping, is an old measure, which finds some theoretical justification in the fact that the blood from the structures behind the spine passes into the same veins as the blood from the spinal cord itself. Hence this measure may conceivably have some influence on the circulation in the cord. If the patient's strength is not such as to render the abstraction of blood desirable, dry cupping may be employed, or the vessels of the skin may be dilated by hot fomentations, or a mustard plaster, or hot water bags. By stimulating the cutaneous nerves, these agents may also influence, in a reflex manner, the vessels of the cord. The application of cold to the spine, as by a spinal ice-bag, has also been recommended. Contrary as these therapeutic agents seem, it is probable that each moderates local inflammation in the same manner, by causing first contraction and then dilatation of the vessels of the inflamed part, and so lessening the tendency to stasis of the blood, on which some effects of inflammation depend. Unless there is reason to suspect hæmorrhage, the application of warmth is the safer and probably, judging from experience, the more effectual. At the very onset of inflammation mild counter-irritation is unquestionably useful, and even a blister may be employed; but when the process has reached a considerable degree, it is very doubtful whether counter-irritation has much influence until the acute stage is over.

In other respects the treatment of acute inflammation of the cord must be guided rather by the nature of the process than by the character of the organ in which it occurs, and the fact that it is an acute local inflammation should be kept in view. A nutritious but unstimulating diet, aperients, and diuretics, are desirable in all cases. If there is constipation a free purgative may be given. Whenever there is evidence of a morbid blood-state, it is important that this should, if possible, be improved, but we have still to learn how most toxæmic states can be neutralised. Nitrous ether may be given as a diuretic with some tincture of digitalis, which tends to render the circulation uniform and to lessen stasis by its influence on the small arteries. The reason for diuresis is that probably no local inflammation occurs that is not associated with a morbid state of the blood which may be to some extent relieved by the action of the kidneys. If any special drug is given, it may be combined with those above named. It is as difficult to ascertain the influence of the drugs which are supposed to exert a special influence on myelitis, as it is in the case of other local inflammations, which have no predetermined degree, and tend to subside when they have reached their height. Ergot was recommended by Brown-Séquard, chiefly on theoretical grounds, and has been extensively employed. In rare cases it has seemed to do good. In cases of hæmorrhagic myelitis it may reasonably be given with greater confidence, or ergotin (3 to 5 gr.) may be injected beneath the skin. Belladonna has also been recommended, but the evidence that it influences the morbid process is not strong. Mercury has been largely employed, given by the mouth and by inunction. The influence of mercury on the inflammation of internal organs does not seem so great as upon that of the fibrous tissues and of the structures that invest organs. Certainly in myelitis its effect is less distinct than it is in many cases of meningitis. Iodide of potassium seems to be no more efficacious than mercury. In cases of transverse myelitis occurring in syphilitic subjects the treatment for syphilis seems to have but little influence on the morbid process. It is true that such treatment is rarely adopted at the very onset of acute myelitis, but after the disease has developed energetic treatment does not seem to modify its subsequent course. This is true also of the subacute disseminated myelitis that occurs in the subjects of syphilis, and might be expected to be more amenable. I have known this form, concurring with syphilitic disease of the cerebral arteries, to develop and run its course to a fatal termination in spite of continuous antisyphilitic treatment, to which the arteritis yielded.

In the general management of a case of myelitis, two points are of extreme importance. One is to avoid, by extreme cleanliness and care, the exciting causes of bedsores. The skin should be most carefully watched, and any indication of deleterious pressure met by a change of position or an alteration of the mode of supporting the part. Cotton wool is very useful for this purpose. If there is a marked tendency to trophic changes the patient should be placed on a water-bed. When there is incontinence of urine, the difficulty of avoiding irritation of the skin is greatly increased. For males, a bed urinal is sometimes useful, but often it causes irritation and even sloughing of the prepuce, and then does more harm than good. A quantity of boracic or salicylic absorbent cotton wool, changed as often as it becomes saturated, is one of the best means of meeting this difficulty. It must be remembered that the prevention of bedsores is the prevention of one common cause of death. Should offensive sores form, a quantity of picked oakum, placed outside the immediate dressing, is a cheap and most effective means of preserving the air of the room from the fœtor of the sores, and is also a useful substitute for absorbent cotton wool, in the case of the poor, to absorb the urine or receive fæces that are passed unconsciously. Such material is also useful for relieving pressure when a water-bed cannot be obtained. The second point in management is the treatment of retention of urine. If there is either simple retention or overflow incontinence, the bladder must be regularly emptied by the catheter, great care being exercised to prevent the introduction of contaminating germs. The importance of daily examining the abdomen to see that retention has not occurred, cannot be exaggerated. If the bladder is left full and the urine allowed to dribble away, inflammation is sure to be set up, and probably also pyelo-nephritis. If cystitis occurs, antiseptic washes must be used to lessen, as far as possible, the decomposition of the urine. Under this influence the cystitis usually lessens, and one grave danger to life is obviated.

When the disease of the cord has become stationary, the patient may be allowed to move, and a more tonic treatment may be adopted. Iron, quinine, or arsenic may be given. Strychnia must be given only in very small doses if there is any excess of reflex action. Occasional counter-irritation may be employed, repeated frequently if any improvement seems to result. The limbs may be rubbed, and any muscular wasting treated with electricity. It is not desirable to use electricity as a therapeutic agent while the cord disease is in an acutely active stage. There is no evidence that the application of electricity to the spinal column has any influence on the process of recovery of the cord. Its chief value is to maintain, as far as possible, the nutrition of any muscles of which the nerves have undergone degeneration. In cases of dorsal myelitis, in which the legs are well nourished, and the reflex action is excessive, it is better not to apply any form of electricity. The unavoidable stimulation of the sensory nerves tends to increase the reflex over-action. Careful attention should, in all cases, be paid to the position of the limbs during the stage of helplessness, so as to avoid as far as possible the development of contractions. For the condition of active spasm, which often develops after severe myelitis, not much can be done; such special treatment as can be adopted is described in the chapters on Primary Spastic Paraplegia.

ABSCESS OF THE SPINAL CORD.

Simple inflammation of the spinal cord scarcely ever goes on to the formation of pus, although leucocytes may accumulate at certain points of the grey matter so densely as to constitute microscopic collections of pus, and in very rare cases of this kind such minute abscesses have been sufficiently large to be visible to the naked eye. Pus only forms in the substance of the cord in considerable quantity in cases of purulent meningitis. In most instances the purulent meningitis has been of septic origin, rarely traumatic. In the former case, suppuration within the brain may coincide with that in the spinal cord, and in the latter it may be very extensive and occur at more than one spot. The symptoms are those of an acute irritative myelitis, but they are often lost in those of the purulent meningitis which precedes the disease of the cord itself. Their special feature is their association with a cause of septic suppuration, as well as with a high temperature and other symptoms of a septic blood-state.

A good example of the disease is a case recorded by Nothnagel. A patient suffering from cough and most offensive expectoration was suddenly seized with severe pains on both sides of the abdomen, attended by a sense of constriction and quickly followed by paralysis of the bladder and of the legs, with loss of sensation and of reflex action. An abscess of the cord was diagnosed. After death there was extensive purulent spinal meningitis, and the dorsal and lumbar cord contained an extensive collection of gangrenous offensive pus, which seemed to occupy the central part of the cord, from the cervical enlargement downwards. Some abscesses were found also in the white substance of the brain. In another case described by Ullmann[*] two extensive foci of suppuration existed in the cord, one cervical, the other lumbar; the former had caused extensive destruction of tissue, and the pus had escaped into the subdural space. The affection was supposed to be secondary to gonorrhœa.

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[*] 'Zeitschr. f. kl. Med.,' xvi, 1889.

EMBOLISM OF THE SPINAL CORD.

The occurrence of embolism in the spinal cord has not yet been proved, but a few cases have been met with which suggest the possibility that the process has been the exciting cause of an acute myelitis. In a young man with mitral regurgitation, considerable weakness of the right leg came on suddenly—in a moment—with transient spasm. The onset indicated a sudden lesion in the cord, which might well have been the embolic obstruction of a small vessel. In a case recorded by Weiss, a boy aged sixteen, with chronic mitral disease, was suddenly seized with complete paraplegia, followed by bedsores, &c. He died four months after the onset, and the lumbar enlargement was found completely softened, with old coagula in the arteries. There was embolism of the kidneys and spleen, and the cortex of each cerebral hemisphere presented small foci of softening. Such cases justify a suspicion that embolism may be the cause of a sudden lesion of the cord in a patient in whom a source of embolism exists, and the process has occurred in other organs.

CHRONIC MYELITIS.

The spinal cord may be the seat of chronic inflammation, which develops slowly, as such, in the course of a few or many months; and the condition may also occur as the sequel to acute myelitis, which instead of subsiding, may persist, manifesting from time to time signs of activity. It is often difficult to say whether such a condition should be regarded as an acute myelitis that has not subsided or a chronic inflammation beginning acutely. In many instances, indeed, it is probable that the disease is most accurately regarded as a combined form in which the causes of both are operative, and a subsiding acute inflammation is arrested and maintained by the influences that would be capable of inducing a primary chronic myelitis. Such progressive tendency is especially conspicuous in the disseminated form, in which the foci of inflammation may remain and fresh ones may develop from time to time in a chronic or subacute manner.

The lesions of chronic myelitis resemble those of acute myelitis in seat and distribution, and intermediate cases connect them by analogous subacute forms. They differ from acute inflammation both in the longer time occupied in their development, and in the absence of the considerable vascular disturbance which forms part of the acute process. Such chronic myelitis may be focal, disseminated, or diffuse. In the former case it may involve the whole thickness of the cord at a certain level, *chronic transverse myelitis*, or only part of it, sometimes one half, occasionally for a considerable vertical extent. The *chronic* *disseminated myelitis* may resemble the corresponding subacute form in distribution, many points of inflammation being scattered through a small region, or through a large part of the cord, and its symptoms may become diffuse by the union of the effects of the disease.

The term "chronic myelitis" has, however, been used in a much wider sense, and has even been applied by some to all local processes attended by an increase of interstitial tissue, whether this is primary or is merely secondary to a degeneration of the nerve-elements. Thus the degeneration of the nerve-cells in progressive muscular atrophy has been regarded and described by many as a chronic myelitis of the grey substance, notwithstanding the fact that there is usually a degeneration of the whole motor path, in both segments, from the cortex of the brain, through the pyramidal tracts and motor nerves, and that the anterior cornua merely constitute, as it were, a focus in which the effects of the processes are especially conspicuous. Such a morbid process is evidently quite different in nature from a focus of ordinary inflammation, in which the primary change is interstitial and random in range and effects, and presents no limitation to function in the incidence of the consequences on the nerve-structures. Whenever this latter feature can be traced, the malady must be regarded as a "system disease," *i. e.* as a disease affecting a system of nerve-structures that have a common function. If "system diseases" are forms of inflammation, they are "parenchymatous inflammations;" and those that are chronic in course from beginning to end are more commonly termed "degenerations," and are so classed and described in these pages. Only then the chronic interstitial processes of random position and influence are here considered.

Etiology.—The causal relations of chronic myelitis, even as thus limited in conception, are very various. An inherited neuropathic tendency can sometimes be traced, but far more rarely than if the cases are included in which the nerve-elements suffer primarily. The disease is most common in early and middle adult life, but it occurs occasionally in old age; it is met with in both sexes, and is perhaps more prevalent in females in the first half of middle life and in males in the second—an important fact, because so many of the cases in young adult women are thought to be hysterical in nature.

Various conditions which lessen constitutional strength predispose to the disease. Definite causes often cannot be traced, although many of the exciting causes of acute myelitis seem capable of giving rise to the chronic form, when acting for a considerable time, or frequently repeated, or in specially predisposed persons. Thus while a single intense exposure to cold may produce acute myelitis, frequent habitual exposure may cause the chronic form. Injury is a frequent cause, and seems to act in more than one way. Chronic inflammation may develop in the vicinity of a damaged spot, when the traumatic lesion may have been too slight to cause pronounced symptoms, or may have caused slight effects which are subsequently lost in the more severe and extensive consequences of the myelitis. In other cases the effects of the lesion may be too slight in degree to be at first perceptible, but (either by a slow influence on the processes of nutrition, or by extensive minute interstitial lesions that induce secondary traumatic inflammation) the symptoms of extensive damage to the cord slowly follow an injury that has no immediate effect on function. These cases will be further considered in a subsequent chapter. Repeated over-exertion has also seemed effective in some cases. Sexual excess has been thought to be a cause, but on less clear evidence.

Chronic myelitis may be secondary to other local morbid processes, especially to adjacent inflammation, which, when it also causes compression, invariably gives rise to chronic myelitis; the inflammation that results from pressure on the cord may be either chronic or acute.

Another series of causes consists of constitutional states capable of giving rise to local inflammations. Chronic alcoholism is, perhaps, the most influential and important of these. Chronic myelitis due to it is usually associated with chronic meningitis, and most intense at the periphery, beneath the inflamed membranes ; but it thence extends into the substance of the cord, and sometimes tracts of irregular extent and position are involved, without connection with the membranes. This cause is one of especial importance, because it often co-operates with others, and determines effects that may seem to be independent. Its effect is met with, for example, in many cases of chronic myelitis that are excited by injury and seem to be due solely to the excitant, although the results of this would have been trifling and transient but for the tendency induced by the preceding and often profound effect of chronic alcoholism on the tissues. It also complicates many cases of multiple neuritis due to the same cause, in which the effects of the peripheral lesion obscure to a large extent those of the central mischief, by causing symptoms that prevent the recognition of the latter, since its consequences are lost, as it were, in those of the damage to the nerves.

The gouty diathesis is another cause of chronic myelitis. We are only beginning to realise how often it acts on many organs and tissues, and how frequently inflammation is thus induced. It is certain that this is a common cause of inflammation in the substance of organs and in mucous membranes, and chronic, as well as acute myelitis, is among its effects. Many cases are due to this influence, and it is instructive to note that, like chronic alcoholism, it is also a cause of inflammation of the peripheral nerves. The disseminated and progressive forms of chronic myelitis are especially due to it, and the analogy should be noted which these present to the irregular forms of perineuritis, of which this state is also a common cause. It is probable that many cases of chronic myelitis in young adults, at present mysterious in origin, and in the obstinately progressive character they sometimes manifest, will be found to be due to the influence of inherited gout, which is certainly capable of causing analogous inflammation elsewhere in the nervous system, as, for instance, in the optic nerves.

Another cause of chronic myelitis is constitutional syphilis. The forms that are apparently due to this are of two types. (1) A diffuse interstitial inflammation, most intense at the periphery, with much new formation of tissue and thickening of the walls of the vessels, both processes extending into the cord from the pia mater, in which a similar damage exists. This form may be subchronic in course, and presents some histological correspondence to known syphilitic processes. (2) A focal very chronic indurating myelitis, such as shown in Fig. 102, which has no distinctively syphilitic feature, may occur in very late stages of the constitutional disease. In neither form does the influence of treatment afford evidence of the relation to syphilis. In the second, sclerotic variety, it does not appear that treatment has any power to modify the morbid process, and in the first it is evident that any cicatricial process that could be induced would only maintain the damage to the nerve-elements, which would suffer further from the maintained or renewed compression under the prolonged contraction of the enclosing tissue. Yet the occurrence of these forms in the subjects of syphilis, the absence of other causes, and the circumstances that the negative facts are not without analogies, make this causation on the whole the probable one.

Pathological Anatomy.—The morbid appearances vary much in the different forms. In most cases the disease is revealed to the naked eye by a change in the aspect of the affected part, in which the white substance is grey in tint, and the grey matter darker than normal, while both are reduced in size in cases of long duration, so that the whole cord may be lessened at the affected part, and its shape changed when the disease does not involve its entire thickness. But in recent cases, the alteration in tint may be the most conspicuous change, and may be visible on the surface, even through the pia mater; this may be thickened at the spot if the disease reaches the surface. The consistence of the affected area may be lessened or increased. Rarely, in recent cases, there is a slight increase in the size of the affected part. In some recorded cases in which a great increase in size has been described, it is probable that an infiltrating glioma was mistaken for chronic inflammation. When there is one considerable focus of disease there may be other slighter foci in the neighbourhood. In what is called the "diffuse" form, the diseased areas are scattered through a considerable extent of the cord; they may blend into a lesion that is truly diffuse, or may remain, in point of fact, disseminated, but be so placed that their effects blend in what may be termed a diffuse influence. Thus it may be found, post mortem, that there are numerous foci of disease when nothing in the character of the symptoms prepared us for a disseminated lesion.

It is probable, indeed, that this is the rule in the chronic form, and an actually diffuse extensive inflammation is much more rare than is suggested by the symptoms of the disease. In the secondary form, the inflammation is usually confined to the neighbourhood of the morbid process which causes it. That which is secondary to meningitis is most intense in the peripheral part of the cord, but sometimes extends deeply, especially when the cause of the meningitis is one, such as alcoholism, capable also of giving rise to myelitis. In this

Fig. 102.

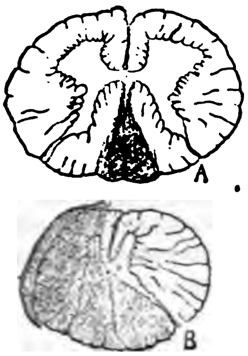

Fig. 103.

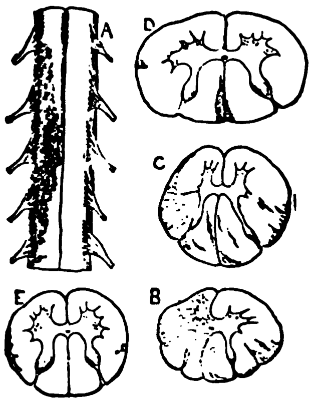

Fig. 102.—Chronic myelitis. (After Charcot and Gombault.) B, section at the level of the third dorsal, shows both the posterior columns and whole of left half inflamed, except the inner part of the anterior column. The affected part was grey and uniform in aspect, vascular, and firmer than normal. Pia mater and arachnoid thickened. A, section from cervical enlargement, showing secondary degeneration in the post.-med. columns. Similar foci existed in the pons, &c.*

Fig. 103.—Chronic sclerotic myelitis. (After Troisier.) A, posterior view of the affected part, lower dorsal region. The diseased areas were grey in aspect and are indicated by shading. B, middle of lesion, showing extensive damage; C, 2½ cm. higher up, slighter damage; D, cervical region ascending degeneration, post.-med. cols., and a spot of disease in lateral column (probably part of the ascending ant.-lat. tract, secondarily degenerated. E, lower part of lesion. (See next figure.)†

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* The patient had had syphilis twenty-one years before. The spinal symptoms slowly developed during fifteen months, and consisted in paralysis with hyperæsthesia on the left side, with loss of sensibility without paralysis in the right, and a band of anæsthesia to pain around the thorax at the level of the lesion, but the mental dulness of the patient prevented accurate observation during the last few months of her life. (Charcot and Gombault, 'Arch. de Phys.,' 1873, vol. vi, p. 143.)

† The patient was a woman forty years of age. The symptoms slowly developed during the three years before death, and consisted of weakness with rigidity in the left leg, and impairment of sensibility in the right, first of the temperature-sense and afterwards of touch. (No observations were made on sensibility for the last fifteen months of the patient's life, during which the lesion was slowly progressing.) (Troisier, 'Arch. de Physiologie,' 1873, vol. v, p. 716, Case 2.)

condition almost the whole thickness of the cord may be involved (cf. A and B, Fig. 105). Now and then, when there is no primary meningitis, the superficial layers of the cord are chiefly damaged, a form that has been termed *chronic annular myelitis* or *annular sclerosis*, because the affection extends like a ring around the cord. This form of lesion is, however, as we have seen, sometimes merely an ascending degeneration of fibres that lie near the surface, and degenerate above the primary lesion.

In chronic myelitis, according to the limited use of the term already explained, the microscope shows that the inflammation is primarily interstitial. The affected areas always stain deeply with carmine in consequence of the great increase in their connective-tissue elements, and may be occupied by a dense fibrous reticulum, in which few nerve-elements can be discovered, as in Fig. 104. In less affected parts of the white columns there is an irregular increase of the interstitial tissue, partly fibrillated, partly amorphous, or studded with nuclei (Fig. 104, upper small figure), while in other parts it may contain many nucleated cells, oval, fusiform, or stellate. The large stellate

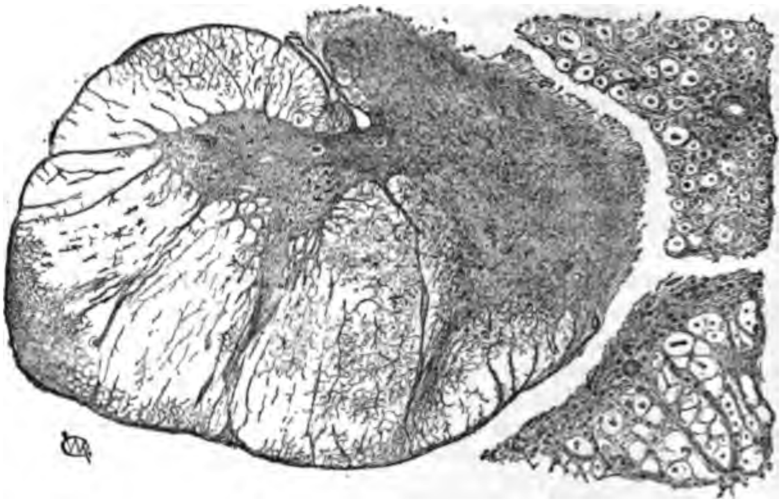

FIG. 104.—Chronic sclerotic myelitis, same case as Fig. 103; section near B, reversed. Almost the whole left half of the cord (right in the figure) is changed into a dense mass of shrunken connective tissue, and the right half is being invaded in many parts by the same process. The upper small figure is from the posterior column, showing a thickened vessel surrounded by nucleated tissue, among which lie nerve-fibres, many of them much smaller than normal, and some with swollen axis-cylinders. The lower small figure is from the right anterior column (left in figure); the pia mater is thickened and contains many nuclear elements; from it thick trabeculæ extend into the white substance and enclose spaces, from many of which the nerve-fibres have perished.*

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* From a section by Prof. Pierret, kindly lent me by Dr. Dreschfeld.

"spider cells" are sometimes very conspicuous, and interstitial fibrous tracts may be traced to their ramifications.

The nerve-fibres waste before the interstitial process, or undergo destructive changes from the beginning. They may at first present the change by which their myelin stains more deeply than normal; afterwards they become narrowed, and the axis-cylinder is often irregularly enlarged. Ultimately many or all the fibres disappear from the affected area. Here and there the destruction of fibres may be out of proportion to the increase in the interstitial tissue, so that thickened and often granular trabeculæ enclose empty spaces. This

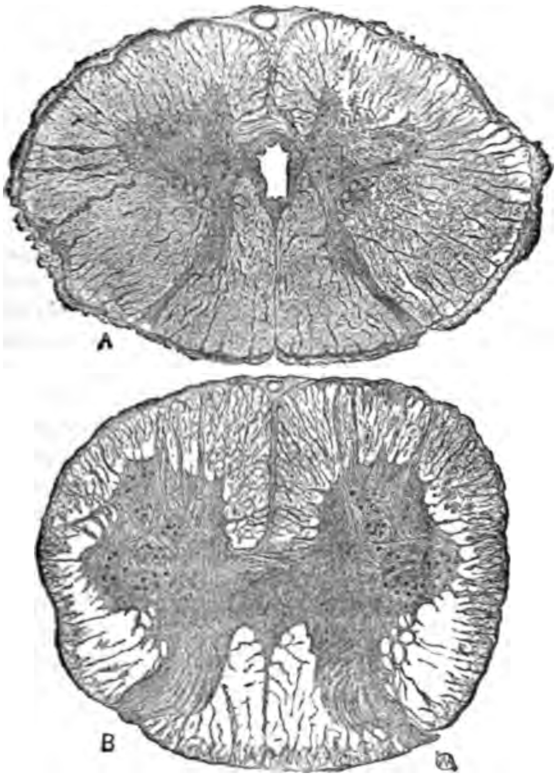

FIG. 105.—Chronic meningo-myelitis, due to alcoholism. Syringo-myelia, congenital, with central gliomatosis. A, lowest part of cervical enlargement; pia mater thickened, and from it tracts of tissue extend into the cord. The greater part of the section is the seat of a diffuse myelitis, in which the nerve-fibres could be seen with difficulty, and were separated by inflammatory products. The grey matter was also affected, and the nerve-cells much changed. The clear spaces in the right ant. cornu appeared occupied by products of degeneration. Central canal greatly dilated and surrounded by new growth, which in B (lumbar section) has obliterated the canal and thus caused its dilatation above. In this section the myelitis is chiefly peripheral, and the grey matter has, for the most part, escaped.

is seen in the lower small illustration in Fig. 104; it is apparently the result of the participation of the nerve-fibres in the inflammatory process during a more general stage, in which all the elements of the tissue were involved. In the recent state, products of degeneration, granule corpuscles, &c., may be found abundantly in the altered regions. The walls of the vessels are thickened, and by this thickening the cavities of the arteries may be ultimately lessened; those of smaller size may even be closed. Not only is the wall of the arteries thickened, but around them the increase of interstitial tissue is often greater than elsewhere, so that the appearance is as if it had spread thence into the adjacent structures. Undue significance has been attached to this condition, especially in old patients with arterial degeneration; it has been regarded as evidence that arterial disease sets up the myelitis, but it is a common appearance in all forms of chronic myelitis, and also in the later stages of acute inflammation. It is probably due to the greater activity of the process in the immediate vicinity of the vessels, and to the share taken by escaped leucocytes in the formation of new interstitial tissue. The peripheral arteries, moreover, occupy tracts of neuroglial and fibrous tissue proceeding from the pia mater, and the increase of interstitial tissue is connected with these; some of it is, on this account, found in the vicinity of the arteries.

In the grey substance there is a similar increase of connective-tissue elements, so that the altered part takes a deeper stain with carmine than the normal grey substance, and has a denser structure, from the greater amount of supporting tissue. The nerve-elements become atrophied; the ganglion-cells, at first swollen and rounder, afterwards shrink, until ultimately they may be reduced to small angular bodies, and they even may disappear. The nuclear corpuscles about the central canal are often increased in quantity, and the canal obliterated, but this condition is so common in otherwise normal cords, that no importance can be attached to it. A considerable increase of tissue about the central canal, closing it below and causing it to be dilated above (Fig. 105, "syringo-myelia"), has been ascribed to chronic inflammation around the canal, but the abnormal tissue is probably always of gliomatous nature, consisting of a development of tissue that is in congenital excess, owing to an arrest of development.

The pia mater is greatly thickened over the inflamed parts, as already stated, and in some cases the affection of the membrane is general, and that of the cord most intense beneath it—a condition termed "chronic meningo-myelitis." It is this form that is shown in Fig. 105. The tracts of new tissue that pass into the cord are dense and wide. As already mentioned, this change may be accompanied by a deeper and sometimes a more diffuse inflammation, as in A, Fig. 105, in which the affection of the cord is greater and that of the membranes less than in B. The chronic myelitis that occurs in syphilitic subjects may present no specific characters. In some cases, however, the cell-formation has been abundant, and has been associated with a tendency to fatty degeneration in the older portions of the lesion and with a similar degenerating hyperplastic inflammation of the pia mater. The condition is, however, clearly one of chronic inflammation, and not of a syphilitic growth.* Nevertheless an actual growth does sometimes coexist with a focus of syphilitic myelitis; it is probable that in such cases the growth is usually the primary change. This may be distinguished by the indications of compression and displacement of the adjacent elements of the cord, a condition which shows that there is an actual new formation.

The more recent the inflammation, the larger is the proportion of cells and nuclei in the interstitial tissue; the longer it has existed, the greater is the amount of fibrillation it presents. The very chronic indurating forms are sometimes called "sclerotic myelitis," or "focal sclerosis of the cord." From the various areas of inflammation secondary degenerations proceed, ascending or descending, and these may complicate the aspect of the disease—a fact which has not always been sufficiently considered in interpreting the morbid appearances.

Symptoms.—The symptoms of chronic myelitis vary much according to the character of the morbid process. For the most part they correspond in character to those that are produced by acute inflammation, differing, however, in their mode of onset. It is therefore not necessary to repeat them in detail. Different cases may manifest almost every symptom that can be produced by disease of the spinal cord. There may be various motor and sensory paralyses, contractures, and even muscular atrophy, but their common feature is their gradual, and often successive development. In the most common form, focal myelitis of the dorsal region, partial or transverse, the symptoms are paraplegic. They differ but little whether there is a single area of disease or several foci near together implicate adjacent structures. Motor power is usually impaired far more than sensibility. Subjective sensations are often prominent and early symptoms, whether there is anæsthesia or not,—tingling, "pins and needles" in the legs, a sensation as if fur or wool covered the skin, sometimes with dull pain in the legs and back, especially after exertion, and commonly also with a well-marked girdle-pain at the level of the chief lesion. Some loss of sensibility, partial in kind or in seat, will often be found if carefully searched for, but there is not often absolute loss of sensation except in the rare cases of intense local sclerotic myelitis, by which conduction may be arrested as completely as by a growth. The onset of the motor weakness is gradual; many months may pass before the power of walking is considerably impaired. As the legs get weak, excess of reflex action is developed (unless the reflex structures are damaged). The knee-jerk is increased; foot-clonus occurs when the legs are in certain positions; and a tendency to spasm gradually develops, and increases to the condition typical of "spastic paraplegia." The sphincters frequently share the impairment of voluntary power. Co-ordination of movement is sometimes affected, usually as general unsteadiness, and defect of equilibrium, rather than as pure ataxy.

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* See Moxon, 'Guy's Hosp. Reports,' 1871, and Charcot and Gombault, 'Arch. de Physiologie,' 1873.

The symptoms may come on simultaneously in the two legs, or one may be affected before the other, and occasionally the paralysis reaches a high degree in one leg while the other still possesses fair strength. When one half of the cord is affected almost alone (as in the lesion shown in Fig. 105), there may even be a crossed paralysis of motion and sensation.

Chronic polio-myelitis is often regarded as a distinct variety, but much confusion has arisen from the fact. If chronic myelitis develops in the cervical and lumbar enlargement, muscular wasting is usually added to the other symptoms. Similar atrophy occurs also in the widely diffused form of chronic myelitis, in which almost all parts of the cord suffer, and it may, indeed, be the most conspicuous symptom. It is always irregular in distribution in these cases, and it may be combined with other symptoms, palsy and anæsthesia, also irregular in extent and seat. The muscular wasting varies in the rapidity of the development, according to the character of the inflammation, and there are corresponding variations in the relations to it of the loss of power in the muscles and also in the electrical reactions they present. In very chronic cases there is a slow failure of electrical irritability to both currents; in others of more rapid course the voltaic irritability persists longer than the faradic irritability, and there may be a distinct degenerative reaction. The symptoms may commence in the arms or in the legs, but they ultimately become general in most cases, and, according to the place of their commencement, may seem to have an ascending or descending course. Occasionally the disease begins in the dorsal region, and after a time, perhaps years, it spreads to the enlargements, and the muscular wasting is added.

The course of chronic myelitis is usually slow and progressive. The symptoms often increase so gradually as only to attain a considerable degree at the end of two or three years. At any stage the progress of the disease may cease. The chronic course may be varied by occasional more rapid increase of the symptoms, due to subacute or even acute processes in the diseased parts. Every degree of chronicity is met with, and subchronic cases, in which the symptoms develop in the course of a few months, effect a gradation to the acute and subacute forms of myelitis. The duration of the disease varies from six months to ten years or more. Thus in one case the symptoms slowly increased during about seven years, and then became stationary, and the patient died twenty-three years after the onset.

DIAGNOSIS.—The diagnosis of chronic myelitis rests on the slow development of symptoms indicating a random process in the spinal cord, i. e. a process which damages irregularly structures of various functions, and thus cannot be looked on as a "system-disease." The maladies from which it has to be distinguished differ according to the seat and character of the inflammation. Dorsal transverse or focal myelitis may be confounded with compression of the cord, with a tumour in it, and with primary lateral sclerosis. The distinction from pressure rests on the absence of a cause of compression, such as bone disease, —especially the absence of the signs of a morbid process outside the cord, preceding those of damage to the cord itself, *e.g.* the pains that indicate irritation of nerve-roots. A tumour within the spinal cord also causes, in most cases, more irritation of the root-fibres than does chronic myelitis. If myelitis involves one half of the cord much more than the other, the symptoms may closely resemble those of an intra-medullary growth. But a tumour never causes much damage to one half of the cord without interfering considerably with the functions of the other half, and the symptoms in chronic myelitis may be strictly unilateral. The distinction from primary spastic paraplegia, the so-called "primary lateral sclerosis," is often one of some difficulty. The motor state of the legs may be identical in the two diseases; in each there is the same extensor spasm, and in each there is a slow, gradual, and apparently simultaneous onset of the weakness and spasm. The distinction is that in primary spastic paraplegia the symptoms are purely motor; there is no indication that the lesion extends beyond the motor structures. In chronic myelitis, on the other hand, there is such indication, either by the impairment of sensation, or by the presence of a girdle pain. This distinction is sufficient in the majority of cases.

The form that involves the grey matter has to be distinguished chiefly from pachymeningitis and degenerative muscular atrophy. In pachymeningitis, the muscular wasting may be similar, but anæsthesia is usually much more considerable in range and in degree, and there is more pain in the limbs. If there are similar symptoms in both arms and legs, myelitis is far more probable than pachymeningitis, since the chronic inflammation of the membranes is less extensive than that of the cord. From progressive muscular atrophy, the chief difference is in the random distribution of the wasting, the absence of symmetry, and the indications of irregular damage to other structures in the cord, as the occurrence of pain and of loss of sensation. But it must be remembered that, by some authorities, most cases of chronic spinal muscular atrophy are regarded as due to chronic myelitis.

For the distinction from other less common diseases, the reader must be referred to the description of them. Like all other organic diseases, cases without wasting of the muscles, occurring in females, are often regarded as "hysterical paraplegia;" the distinction rests on the points already described. It is, however, one of the diseases in which the least excusable errors in diagnosis occur, and in which their effects are often the most disastrous.

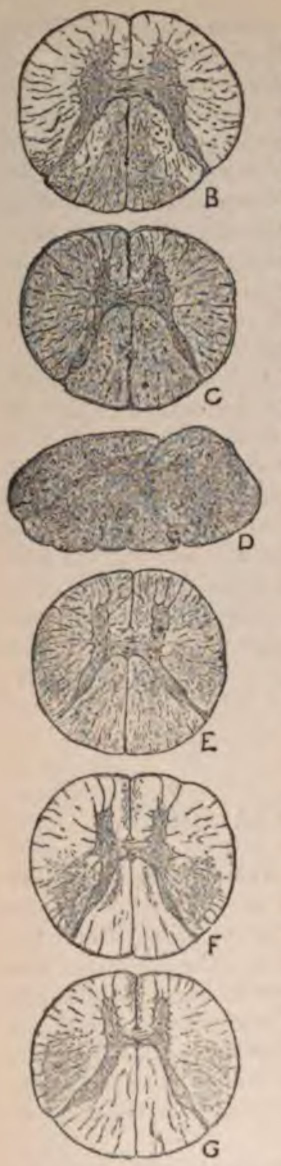
PROGNOSIS.—Chronic myelitis is a very grave disease, on account of the intractability of the morbid process, its frequently progressive tendency, and the persistence of damage which is slowly produced. But cases vary widely, especially in the two former features, and it is a disease in which general prognostic rules can scarcely be formulated. The observed tendency of an individual case is alone a trustworthy guide. Arrest of the disease is often obtained, but actual recovery is rare. The prognosis is little affected by the seat or extent of the process, except that the implication of the grey matter of the enlargements is generally an unfavorable indication. The severity of the lesion, as shown by the degree of the symptoms, is significant chiefly in cases of long duration, and is subordinate to the course and mode of onset. If it can be arrested, the prospect of improvement is better the slower has been the development of the disease, and the shorter the time the symptoms have lasted. In general, moreover, the prospect is better, the fewer the foci of inflammation, in males than in females, and in cases of uniform than of recurrent or relapsing course. Preceding syphilis does not materially modify the prognosis; hence the great importance of the diagnosis from syphilitic growth, in which suitable treatment has a most certain effect. The ultimate danger to life is least in focal myelitis in the dorsal region; it is greatest in the disseminated form, especially in the less chronic cases with a tendency to sloughing of the skin, and in the more chronic forms with muscular wasting. The indications drawn from the invasion of the respiratory muscles, and from the presence of any of the complications that so often terminate life, are the same as in other diseases of the cord.

TREATMENT.—The first and most important measure is the improvement of the general health, by rest, change of air, and tonics. All causes of physical and mental depression must, as far as possible, be removed. Over-exertion, and even fatigue, should be avoided, and the patient should be kept, as far as possible, from exposure to cold. Absolute rest for a short time is often useful at the outset of the treatment, especially when there has been a somewhat rapid development of symptoms. In cases of purely chronic course, absolute rest should not be maintained for more than a few weeks, or the patient may find it hard to regain his former muscular power. Counter-irritation at the situation of the disease is often useful, and most so in cases in which there is spinal pain or tenderness. Repeated sinapisms or blisters may be employed, but a mild form of the actual cautery is on the whole the most useful. A superficial burn or even slight vesication is sufficient on each side of the spine opposite the affected part. It should be repeated as soon as the skin has healed, and if linear cauterisation is adopted the fresh line can be made beside the old one. A hot douche to the back, at a temperature of 103° or 104° F., applied daily, has been strongly recommended by Brown-Séquard. Warm brine baths, and various thermal mineral waters, have been also said to do good. A sea voyage is often of service, combining as it does the maximum of fresh air with the minimum necessity for exertion. Drugs, unfortunately, often fail to influence the morbid process, but nevertheless they are occasionally useful, so as distinctly to justify their careful trial in most cases; and it is important to remember that those that are useless in one stage or period of the disease may yet be of unquestionable service at another stage. Tonics, as quinine and iron, should be given if indicated. Most of the measures recommended, and precautions advised, in the treatment of acute myelitis are needed in the chronic form, and it is therefore superfluous to repeat them. Those drugs that most deserve a trial are arsenic, small doses of mercury (such as $\frac{1}{16}$ gr. of the red iodide), and iodide of iron. Energetic mercurial treatment rarely does good, even when the patient has had syphilis, and sometimes it does harm. Iodide of potassium seems to have little influence on the disease. Nitrate of silver, ergot, and phosphorus have been recommended. Strychnia is chiefly useful in cases in which there is muscular wasting, but is of far less value than in the degenerative muscular atrophy. The treatment of other symptoms is that suitable in primary spastic paraplegia or progressive muscular atrophy, and described in the account of those diseases.

COMPRESSION OF THE SPINAL CORD.

Compression of the spinal cord is a common consequence of various morbid processes. Inflammation is almost always excited by the pressure, and interference with function occurs, partly from the pressure and partly from the resulting myelitis. The symptoms produced have, in different cases, many characters in common, although they vary according to the mechanism of the compression and the acuteness of the inflammation. It is only when there are indications that an acute myelitis has damaged the nerve-elements that we are justified in regarding the interference with function as an effect of the inflammation.

Causes.—The morbid processes that may compress the cord are those that involve an encroachment on, or occupy part of, the vertebral canal. The chief are the following:—Disease of the bones of the spinal column, especially caries; growths in the spine; aneurism eroding the bones and then compressing the cord; growths in the membranes; thickening of the dura mater. These processes have usually only a small vertical extent, and the pressure they exert rarely extends over more than a few inches.



Pathological Anatomy. — The cord usually presents evidence of the compression it has endured in considerable narrowing at the spot, where it may be indented and flattened, or cylindrical. Sometimes the reduction in size is extreme; for an inch or so the cord may be reduced to one third of its normal diameter, and it has even been found no thicker than a crow-quill. An example of flattening is shown in Fig. 106. On the other hand, there is sometimes very little narrowing to be discerned. At the compressed part the cord is usually grey in tint; its consistence is lessened in early cases, and increased in those of long duration. The change in colour and consistence is due to the inflammation of the substance of the cord which always results from pressure, and may often be traced for some distance above and below the compressed part. When there is much compression there is always much inflammation, but considerable myelitis may occur when the amount of compression is slight. The inflammation may be chronic and slow, developing in proportion to the pressure, or it may be subacute or acute, even when the pressure is gradual. The signs of inflammation are very distinct on microscopical examination, and resemble those in other forms of myelitis. There is a general increase in the interstitial tissue; in this, at first,

Fig. 106.—Compression of the spinal cord and pressure-myelitis, in a case of caries of the spine. D, mid-dorsal region, near the chief point of greatest compression; the cord is narrowed from before backwards, and is uniformly damaged by inflammation, so that the grey substance can scarcely be distinguished. C, 1½ inches higher up, shows a slighter degree of myelitis, still extending through the whole thickness of the cord. B, first dorsal; the myelitis is much slighter and the chief disease is in the posterior columns, in which ascending degeneration occupies the post.-median columns and extends outwards into the post.-external columns. E, 1½ inches below the point of greatest pressure; inflammation still extending through all the elements of the cord. F, 2 inches lower down, shows only descending degeneration in the pyramidal tracts, anterior and lateral, and also the "comma-shaped" descending degeneration in the anterior part of the post.-external column. In G, at the lowest part of the dorsal region, the comma-shaped degeneration has ceased, and only that of the pyramidal tracts remains. (From preparations by Dr. F. G. Penrose.)

ious cell-forms may be found, but it ultimately presents the pearance of a dense reticulum. The nerve-elements undergo de-ieration, and abundant masses of myelin, granule corpuscles, and pora amylacea are visible in the fresh state or in glycerine pre-rations of the hardened cord (Fig. 107, c). Many nerve-fibres rsist, however, with a narrowed sheath of myelin, and it is probable at these regain the power of conducting, in spite of the persistence considerable, and even dense sclerosis in the part. In extreme ses all the fibres seem to be destroyed at the point of chief compres-

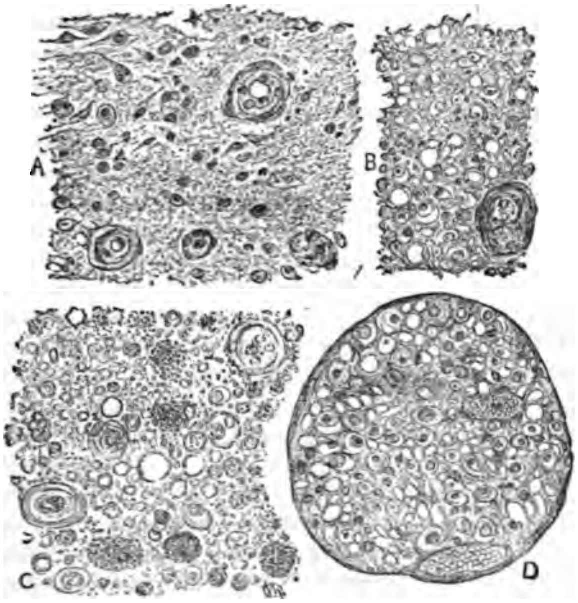


FIG. 107.—Pressure-myelitis; portions of the section D in Fig. 106 more highly magnified. A, from the grey matter, numerous angular and fusiform cells; vessels with walls thickened by a growth of cells, narrowing the cavity, which in some is obliterated. B, from the white columns; thickening of the interstitial tissue, nerve-fibres in part destroyed, in part narrowed; a vessel with thickened walls. C, glycerine preparation from white substance; abundant products of degeneration of the nerve-fibres, in part aggregated into granule-masses. D, section of a nerve-root passing by compressed area; increase of interstitial tissue, many nerve-fibres narrowed, some with swollen axis-cylinders.

on, but there is never an actual division of the cord itself. The grey atter can scarcely be distinguished from the white, even with the icroscope, and the ganglion-cells become shrunken and atrophied. he walls of the small vessels are often thickened by spindle-cells, isposed more or less concentrically to the cavity, which is encroached pon and may be obliterated (Fig. 107, A, B), a process that must add the degree of damage to the cord. The signs of inflammation radually lessen above and below the compressed part, but often extend for some inches in each direction. Beyond its limits the usual ascending and descending degenerations may be traced (Fig. 106). The nerve-roots passing by the seat of compression suffer from the pressure, and from interstitial inflammation excited in them. They are usually grey in tint and ultimately waste, and may even be reduced to fibrous threads. The microscope shows increase of the interstitial tissue, wasting of many nerve-fibres (Fig. 107, D), and enlargement of some axis-cylinders.

SYMPTOMS.—The effects vary much according to the degree of pressure, its rapidity, its direction, the amount and character of the inflammation produced, the amount of damage to the nerve-roots, and the position of the disease. The symptoms in most cases enable the fact of slow compression to be inferred, even when there is no indication of the cause. They are of two classes: (1) interference with the function of the nerve-roots at the level of the morbid process; (2) interference with the functions of the cord itself. The cord symptoms consist chiefly in impaired conduction, manifested in the parts below the lesion. The central functions of the cord (reflex action, &c.) at the level of the lesion are abolished by the pressure, but the symptoms of this abolition are often lost in those of the damage to the nerve-roots.

Of the root symptoms the most constant is pain, extending along the course of the nerve-fibres, and through the area of their distribution. The seat of these pains depends on the position of the disease; they may be felt in the arms, around the thorax or abdomen, or in the legs. They are usually sharp pains, resembling neuralgia in character, sometimes accompanied by tender points. Occasionally, when felt in the limbs, they are referred especially to the joints. The pain may be intermitting or constant. It may exist alone, for a long time, in a disease that increases slowly, and sometimes may be the only symptom, although some compression of the cord occurs. With it there is usually hyperæsthesia of the skin, often intense. After a time anæsthesia develops in irregular areas, in spite of the persistence of the pain,—the condition termed "*anæsthesia dolorosa.*" Irritation of the motor nerve-roots may cause painful contracture of the muscles, but this is, on the whole, rare, and the chief motor symptoms are due to the interruption of the fibres—muscular weakness, gradual in onset, and accompanied by wasting. The rapidity of the atrophy varies much, and with it the electrical reaction. When slow, there may be merely a progressive diminution in irritability to faradism and voltaism; when rapid, there is often the degenerative reaction, and sometimes the "mixed form," from the partial damage to the fibres that supply a muscle.

These local root symptoms are usually the first, and to them are added, after a time, the indications of interference with the function of the cord itself. There is weakness in the parts supplied from the cord below the lesion. The loss of power is usually gradual, but sometimes comes on rapidly, even in the course of a few hours, when an acute myelitis is set up by the compression. With the weakness there is a marked and early increase in the superficial reflex action, more constantly conspicuous than in most other diseases of the cord, and therefore of some diagnostic importance. The myotatic irritability is also increased, a foot-clonus being almost always present. Pains in the legs may occur, even when the disease is above the lumbar enlargement, usually dull and aching. There is often formication and tingling. There may be no impairment of sensibility in the parts below the lesion, even when there is complete motor palsy; in many cases, however, there is some sensory loss, complete only when the spinal lesion is very severe. Conduction of painful impressions may be delayed, sometimes for thirty or even forty seconds. When the pressure is lateral, one leg may be first and most affected, but the other side is usually soon involved, since neither the mechanical effect nor the inflammation remains limited to one side of the cord. The sphincters are often affected. The tendency to the formation of bedsores is rarely great, unless the lumbar enlargement is compressed or myelitis descends into it.

The course of the symptoms varies according to that of their cause. When an acute myelitis has been set up, improvement may occur for a time even if the cause of pressure is slowly progressive. This is true also of root symptoms when they are rapidly developed. If the compression ceases to increase, the cord may recover its conducting function, although it remains narrowed. Sensation is often regained in the legs when motion remains absent, but both motion and sensation may be regained although the narrowing of the cord persists. It has even been found no larger than a goose-quill at the compressed spot, although the paraplegia had passed away. In such a case many narrowed nerve-fibres are found in the section of the compressed portion, and it is probable that many axis-cylinders persist, with an envelope of myelin so narrow as to be recognised with difficulty, but which suffices for their conducting function. Under slow pressure the axis-cylinders may not suffer interruption, although the myelin disappears. Regeneration of axis-cylinders, as in the peripheral nerves, is also possible. In some cases, especially when a considerable myelitis has been set up, the motor path may recover, but the posterior columns remain affected, so that power returns, but without co-ordination,—"secondary ataxy" resulting.

The DIAGNOSIS of slow compression rests on the root and cord symptoms already described, and especially on their coincidence with a cause of compression. Of the former, the sensory symptoms are the most characteristic. In many cases other indications of the compressing disease assist the diagnosis. These vary in character; the most frequent is considerable local tenderness of the spine. If root symptoms are absent, the diagnosis is much more difficult, unless the morbid process manifests itself externally. If there is a suspicion of compression in a case of slow paraplegia, an early and considerable excess of superficial reflex action gives additional weight to the suspicion. Even without any other symptoms to indicate compression, this is suggested by symptoms which begin on one side and gradually spread to the other.

The chief disease from which compression of the spinal cord has to be distinguished is a subacute or subchronic transverse myelitis. Besides the indications of a cause of compression already mentioned, the chief distinction depends on the presence of symptoms indicating irritation of the nerve-roots, and especially on the fact that these symptoms precede those of the lesion of the spinal cord itself. Although a very chronic myelitis is occasionally attended by much irritation of the nerve-roots, the symptoms of this succeed, instead of preceding, the damage to the cord, and the course of chronic myelitis is often much slower than is that of most forms of slow compression. The distinction from a growth within the spinal cord rests chiefly on the early occurrence of impairment of the central and conducting functions of the cord, and also on the partial distribution, and often slower course, of the symptoms. The effects of an intra-medullary growth vary widely in different cases, and if there is an early irritation of root-fibres, the distinction may have to depend on the presence or absence of the signs of an external compressing process.

The diagnosis of the cause of compression is often much more difficult than that of its occurrence. The facts that the patient is in the first half of life, and that he inherits a tubercular tendency, suggest caries. Slightness of root symptoms is also in favour of caries or a tumour within the spinal cord; the presence of these symptoms does not, however, render caries less likely, unless the pain is extremely severe and is greatly increased by movement,—characters that should always suggest a growth in the bones of the spine. Root symptoms extending over a considerable vertical area suggest pachymeningitis. Additional details of the differential diagnosis will be found in the account of the several compressing diseases.

Prognosis and Treatment.—The prognosis depends on the cause, and the amenability of this to treatment. As a general rule, however, the more rapidly the cord symptoms develop, the more chance there is of improvement, because the greater share does inflammation take in their production. The treatment is that of the cause of the compression and that of myelitis.

ANTERIOR POLIO-MYELITIS; ATROPHIC SPINAL PARALYSIS.

In certain diseases of the spinal cord, wasting of the muscles is a prominent symptom. In these the lesion involves, exclusively or in part, the anterior grey cornua, and the muscular atrophy depends on the destruction of the nerve-cells, from which the motor nerve-fibres proceed, and on the consequent degeneration of the fibres. The muscular wasting does not depend on the nature of the lesion; it occurs equally whether the nerve-cells are destroyed by hæmorrhage, by inflammation, acute or chronic, by slow degeneration, or by pressure. But the rapidity with which the destruction occurs influences the character of the symptom. Time is required for the change in the nutrition of the muscles; one or two weeks elapse, even in the most acute cases, before wasting is distinct. The nerve-cells also form part of the voluntary motor path, and their disease at once interrupts this; hence, in acute cases, paralysis is rapidly developed, and the atrophy succeeds it after an interval. In chronic lesions, on the other hand, the changes in the cells and muscles progress so slowly that the weakness and wasting appear to come on together. The wasting may even seem to precede the weakness, but a careful examination will show that power is defective as soon as there is distinct atrophy. It is customary to separate the two groups by terming those in which the palsy precedes wasting, "atrophic spinal paralysis;" and those in which wasting is apparently simultaneous with weakness, "spinal muscular atrophy."

The atrophic paralyses are due to an acute or subacute process in the anterior cornua. The lesion is probably, in most cases, inflammatory in character, a cornual myelitis, or "polio-myelitis" (Kussmaul). In some cases, which differ chiefly in the suddenness of the onset, it is probable that the lesion is hæmorrhage and not inflammation.

The chronic atrophies are due to a slow degeneration of the cells and fibres. Between the two forms, however, we meet with others of intermediate course, subacute or subchronic, due to processes of corresponding character,—usually inflammation, interstitial or parenchymatous.

ACUTE ATROPHIC PARALYSIS; ACUTE ANTERIOR POLIO-MYELITIS.

Infantile Paralysis; Essential Paralysis of Children.

Acute atrophic paralysis is a disease in which voluntary power is lost in the course of a few hours or days, and in which some of the paralysed muscles undergo rapid wasting while others recover. The onset of the palsy is often preceded or accompanied by indications of general disturbance. The muscles which waste usually remain weak, and contraction of their opponents leads to permanent distortion of the paralysed limbs. Although it is usually a form of acute myelitis, its characters and course are sufficiently special to make its separate consideration desirable. The disease is the most common form of paralysis in young children, and has hence received the name of "infantile paralysis." Before anything was known of the lesion on which it depends, it received (from Rilliet and Barthez) the unmeaning designation of the "essential paralysis of children," a term that is falling into merited disuse. The spinal lesion was first demonstrated by Prevost (1865), and soon afterwards by Lockhart Clarke and by Charcot and Joffroy. Its constancy has been abundantly proved by numerous observations during the last twenty years. The occurrence of similar symptoms in adults was noted by Vogt (1858) and Duchenne (1864).

ETIOLOGY.—The disease occurs at all ages, but is ten times as frequent in the first decade as in all the rest of life. It is, moreover, especially a disease of later infancy. Of the cases in early life, three fifths occur in the first three years of life, nearly one fifth in each year. The numbers in each successive year (of 116 cases of which I have notes) were—in the first 21, the second 21, third 25, fourth 9, fifth 17, sixth 4, seventh 2, eighth 6, ninth 4. Of the cases that begin during the first year, nearly all begin in the second six months; the earliest of the series occurred at four months, but one at the twelfth day is described by Duchenne. The friends often date the disease from birth, but there is no real evidence of its intra-uterine development. After childhood it is most frequent between ten and twenty, and very rare over forty. Many supposed subacute cases in adults have been examples of multiple neuritis. In childhood the two sexes suffer equally (males 59, females 57), but after ten the malady is practically confined to the male sex. The influence of heredity is small, but a family tendency to suffer may now and then be traced; two and even three brothers or sisters have been affected (Seeligmuller, &c.). One of my own patients had a sister, and another two cousins, affected with the same disease. The inherited tendency may be indirect; of a patient in whom the disease came on in adult life, two brothers had suffered from hemiplegia, and an uncle from paraplegia.

The disease presents a remarkable relation to season, occurring far more frequently in summer than in winter. Sinkler, who first called attention to the fact, found that in Philadelphia four fifths of the cases commenced in the five hot months, May to September. In this country the relation is equally apparent; three quarters of the cases occur during the hottest third of the year, June to September. The distribution of 71 cases was as follows: One occurred in each of the first five months; in June, 11; July, 13; Aug., 13; Sept., 15; Oct., 6; Nov., 2; Dec., 5. In many cases, the weather was very hot at the time; in some, however, the disease came on in cooler weather after a hot period. It would, therefore, seem that the heat generally acts as a remote cause, and is only occasionally concerned directly in the exciting mechanism. The fact is the more important because cold has long been regarded as the most potent exciting cause. That exposure to cold is an occasional cause is certain, although the number of cases in which it can be traced is not large; and it is usually a general and not a local exposure, and has not often been immediately preceded by exposure to heat. Exceptions are chiefly met with in adults. Thus one patient, aged sixteen, when perspiring from a long ride after the hounds on a hot day in September, lay down on a sofa beneath an open window from which a draught blew on his back. Two days afterwards the first symptoms commenced. I have twice known the disease to be due to sitting on damp grass. Two or three days is the interval which usually elapses between the exposure to cold and the onset of the symptoms, but great variations are met with, and the constitutional disturbance may commence within twenty-four hours of the exposure.

Over-exertion in walking seems occasionally to aid in exciting the disease; in some cases the child has walked far more than was proper the day before the onset, and in others habitual over-exertion has been permitted. A traumatic influence, such as a fall, occasionally precedes the onset in such a way as to make its influence probable. Thus, in one severe case, a boy eight years old was thrown over the head of a donkey, and the onset occurred five days afterwards. In most cases the fall has produced no immediate symptoms, and has perhaps acted generally rather than locally. Thus a girl of four fell off a chair, striking her head, and was stunned for a moment; two days later she was sick and feverish; the temperature (101°) continued raised till the sixth day, when general loss of power was found, and the residual state was atrophic palsy of both legs and the right deltoid. The cases following falls and over-exertion present the same relation to season as do the rest.

There is no proof of any relation between the disease and the process of dentition. The period of dentition coincides with the rapid functional development of the nervous system which succeeds its structural development in the first few months of life. Moreover, this period is often one of deterioration of health, in consequence of changes of diet and other causes. The disease occurs before and after the period of teething, and it exhibits no increase in frequency at the period of the second dentition.

Many children are perfectly well at the time when the disease occurs. A few are in conspicuously defective health. Thus in one case the child had been rendered feeble by long-continued diarrhœa. I have only once seen the disease in the subject of inherited syphilis. It has been thought to be occasionally secondary to acute febrile diseases, but the frequency of the relation has been unquestionably overrated; in some supposed cases in adults there has been multiple neuritis only, and in children initial general disturbance is constantly mistaken for an independent general disease, and the opinion is often maintained after the discovery of the paralysis, which is then supposed to be of secondary origin. Thus in one case initial pyrexia, headache, and vomiting were supposed to indicate scarlet fever. Paralysis of all four limbs came on, with difficulty in swallowing, and the latter was supposed to render certain the diagnosis of scarlet fever, although there was no sore throat or rash. In older children and adults the disease is often thought to be secondary to rheumatic fever, in consequence of the rheumatoid character of the pains. The error is the more likely to occur if the symptoms follow exposure to cold. It is conceivable that catarrhal affections may be produced by the exposure to cold that induces the disease; but of the 116 cases referred to above, not one was related to any acute specific disease, or to rheumatic fever. Chronic alcoholism has been thought to be a cause in adults, but it is probable that most cases supposed to be due to this influence have been instances of multiple neuritis.

Symptoms.—The general characters of the symptoms have been already mentioned—an acute onset, often with constitutional disturbance; paralysis, at first wide-spread, afterwards passing away, except from a region in which the muscles rapidly atrophy, and in which, although partial recovery may occur, more or less weakness and wasting persist.

The general disturbance, which is apparently due to a morbid blood-state, may be severe or slight. In most cases there is pyrexia, with its usual accompaniments, headache, prostration, loss of appetite, restlessness. Vomiting is very common, and often causes the illness to be mistaken for stomach disturbance. Occasionally there is also diarrhœa. In children, vomiting is very common, and generally causes the illness to be mistaken for simple stomach disturbance. These symptoms of general disturbance usually last for a few days, sometimes for only a few hours, and they are occasionally, but not often, absent. The palsy develops in most cases in the course of this disturbance. A common history is for a child to seem and feverish, to be put to bed, and the next morning to be found have lost power. In other cases the febrile disturbance lasts several days before the paralysis comes on. But the relation of t general disturbance to the onset of the paralysis presents gre variations, which appear (from a comparison of cases) to be indep dent of any variations in, or special features of, the spinal sympto The constitutional symptoms occasionally succeed the onset. Th a child, well one day, was found next morning to have both l paralysed and was feverish and sick; the general disturbance c tinued for a week. The elevation of temperature is usually moder in degree; an example of its character, in what may be regarded a fairly typical case, is that of a child whose temperature on second day of illness was 101·2°; on the third, 101°; the fourth which the palsy developed), 100°; the fifth, 99·5°; the sixth, It is usually, as in this case, highest at the commencement, and sometimes reaches 103°, 104°, or even in rare cases 105°. The constitutional symptoms are present in most of the cases in which a fall appears to excite the disease, and are of the usual character; they are as marked in the cases that appear to be spontaneous as in those that follow exposure to cold.

Convulsions occasionally attend the onset, chiefly in young children. In some instances, moreover, the initial symptoms are those of severe cerebral disturbance. Thus one child had repeated general convulsions during 30 hours, and afterwards was in a state of deep coma for 36 hours, the temperature at the onset being 105°. This was succeeded by slighter pyrexia and prostration for three weeks, and it was only at the end of that time that general loss of power was found to have come on. The arms recovered, but atrophic palsy persisted in the legs. In another case, a child of seven months became ill, febrile (102 8°) and comatose with pin-point pupils; it remained in that state for four or five days, and then gradually recovered, but with general powerlessness, rigidity of the legs, and tenderness of legs and back. The rigidity and pain passed away, but the right deltoid and, to a less degree, the biceps and triceps and the whole of the right leg remained paralysed, and presented characteristic atrophy. In adults there is sometimes marked somnolence before the onset, and occasionally slight cerebral symptoms, such as diplopia, giddiness, or (very seldom) delirium. A medical student found, one day, that he saw double; on the next day he felt giddy, and became feverish (102°); he slept for forty-eight hours, and on the fourth day found his right arm was weak; atrophy of the deltoid, &c., followed. The cerebral symptoms always pass away together with the indications of general disturbance, and hence are probably due to the blood-state. In rare cases, the onset is accompanied by symptoms of slight transient spinal meningitis, which must be regarded as a coincident effect of the cause, since there is no evidence in these cases that the myelitis reaches the surface of the cord, and the membranes may be apparently affected early. Thus a child, after being feverish and vomiting for two days, became universally rigid, so that strychnine poisoning was thought of. The rigidity passed away two days later, when the right arm and leg were found to be paralysed. Another child presented all the symptoms of spinal meningitis a week after the onset of paralysis of the arm, and as they passed off the leg also was found affected. When the general illness is severe, the paralysis is often not discovered for some time, especially in a child; the immobility of paralysis is often mistaken for the inertia of prostration.

In older children and adults, another common initial symptom is the pain already mentioned, sometimes referred to the muscles, sometimes to the limbs generally, occasionally to the course of the nerves. It is often absent, but may be very severe and accompanied by extreme tenderness of the limbs. In such cases the pain may be supposed to be in the joints, because it is produced by moving them, when it is really in the nerves, which are mechanically disturbed by the movement of the joints. In these cases there is neuritis, which may come on at the same time as the palsy, although independent of it. Pain may be felt also in the back. In character it is dull and aching. It may be severe, especially when there are symptoms of spinal meningitis. Instead of being initial, the pain may succeed the onset of paralysis, and it occasionally continues for several weeks, especially when seated in the nerve-trunks. In cases of paralysis of the thighs there is sometimes persistent pain on movement at one hip or groin, the cause of which is not clear. Tingling or formication in the limbs may be described by older patients.

The paralysis is almost always rapidly developed. It varies much in its initial range. Only part of a limb may be affected, or there may be universal loss of power; but in the majority of cases the paralysis is intermediate between these extremes. Two or three limbs are affected—both arms, both legs, or the legs and one arm. When all four limbs are paralysed the neck-muscles also may be weak, and even swallowing may be impaired. The other muscles supplied by the cranial nerves escape, as a rule, and the sphincters are unaffected. Occasionally there is incontinence of urine, chiefly in severe cases, and it may then last a long time. For instance, a child aged two and a half years woke up one morning with headache, fever, and weakness of the legs, which rapidly increased to complete paralysis. Four days later the arms also became weak, and in a day or two more the urine escaped involuntarily. The arms began to recover in six weeks, and were well in six months; both legs wasted and remained permanently paralysed; the incontinence of urine lasted for a year. In another case, with incontinence for a few weeks, the residual palsy was in the lower legs. As in these instances, the symptom is met with chiefly when the lumbar region is diseased.

The paralysis may commence in one limb and quickly spread, reaching its maximum extent in a few hours to a few days, but very often the actual onset is so rapid or so escapes careful observation in consequence of the general illness that all the parts first affected are found to be paralysed when it is discovered. Occasionally the onset is in two distinct stages, separated by a few days; now and then the loss of power develops slowly in the course of one or even two weeks, in cases that must be regarded as "subacute," but do not differ in other respects from the ordinary form. Examples o the common onset have been already given; as another may be me tioned the case of a boy, aged a year and four months, who one d seemed ill, was sick, and was put to bed. The next day he could scarce stand alone; on the following day he could neither move the legs n sit up in bed. In about ten days the left leg began to improve, b there was enduring paralysis and wasting of the whole of the rig and of the lower part of the left, leg. In another case, a child seemed ill, and was kept in bed for five days; on the third day it was noticed that the left arm was not moved so much as the right, and by the fifth day the arm was quite powerless; at the end of a fortnight improvement commenced. In the adult, the mode of onset presents nearly the same characters. A lady, twenty-five years of age, sat down for some time on wet grass. Two days later general rheumatic pains came on, very severe in the legs on the following day, and the legs were distinctly weak. In the course of the next forty-eight hours the arms also became feeble, and at the end of a week she could scarcely move her arms, and her legs were absolutely paralysed. In a fortnight improvement commenced, first in the arms and then in the feet; but the hip- and thigh-muscles remained paralysed and rapidly wasted.

Cases are sometimes seen similar in other respects to those now under consideration, but in which the paralysis comes on suddenly, and without general disturbance. Thus a child, aged two, was walking along, when he suddenly could not stand, and fell down on his knees; both legs were powerless. Slow improvement followed in the left leg, and wasting in the right. The suddenness of the onset makes it probable that the lesion is hæmorrhage, and not simple inflammation. Spinal hæmorrhage is known to occur in early childhood. Hence, while we must class these with the other acute spinal atrophies, we are not justified in regarding them as cases of polio-myelitis, since we do not know whether the hæmorrhage is secondary to commencing inflammation or is primary. It is probable however, that even a primary extravasation into the grey matter is due to the same causes that give rise to inflammation, because the two sets of cases occur under the same general conditions of onset, and hence they cannot be separated. Moreover, constitutional disturbance is more often absent in cases of actually sudden onset than in others, although it is sometimes considerable. Thus a child had been ill for two weeks, when, being able to walk about, it suddenly fell to the ground; the left leg was found powerless, and the right weak. It is probable that a sudden onset is more frequent than is suggested by the facts that can be ascertained, since the palsy often comes on during the night or during rest; for instance, in one case a child sat on a chair for an hour, when the left leg was found powerless.

In some cases of sudden onset the initial palsy is universal, and in such cases as the following it is probable that a limited hæmorrhage in the cervical region causes rapid pressure on all the adjacent structures, which afterwards recover from the effects of the pressure. A girl, aged seventeen, suddenly felt tingling in the left hand. In a few minutes the whole arm was powerless, and then the right arm became paralysed. She had a strange sensation at the back of the neck, and went upstairs; as she was going up, her legs became weak, first the left and then the right. In less than half an hour from the time of the first symptom, there was absolute universal paralysis, with difficulty in breathing and in swallowing. Improvement (in the opposite order to the onset) commenced the same evening and was complete in about six weeks, with the exception of the muscles of the left forearm and hand, which rapidly wasted and were permanently paralysed and atrophied. Another girl, while walking across a road, suddenly felt a "sort of shock" as if someone had given her a knock between the shoulders. She became giddy, and instantly felt tingling in both arms, especially in the right, which became weak before she had got to the other side of the road. Ultimately the arms recovered, but permanent paralysis and wasting of the intrinsic muscles of each hand were left.

The initial loss of power is as a rule far more extensive and severe than the permanent affection; in a small number of cases the two correspond. But the extent of the early palsy, that of the enduring atrophy, and the relation between the two, vary extremely, so that no general or even common laws can be discovered. Indeed, in all features of the disease variability prevails to such an extent as to baffle attempts to describe them in general terms, and a better conception of the malady can be gained from such illustrative instances as are here given, than from general descriptions. One general fact is unfortunately true of almost all cases, namely, that the palsy scarcely ever passes away entirely, and that where wasting occurs, wasting remains.

After the paralysis has reached its height and ceased to increase or spread, whether the point is attained suddenly or rapidly or slowly, it remains stationary for a time, which varies from a few days to six weeks, and then lessens. The improvement occurs first in the parts last affected, and gradually spreads until, usually at the end of from one to three months, all parts have recovered except those which are to be permanently affected. In these the muscles are toneless and flaccid from the first, and in two or three weeks there is distinct wasting, which rapidly increases, until the shape of the limb is changed, and, in extreme cases, scarcely any of the volume of the muscle can ultimately be detected. In fat children the appearance of the limb may be less altered, and it seems that, in some of these, an interstitial growth of fat makes up for the diminution of muscular tissue. In older children and adults the muscles are often tender to the touch during the process of wasting.

When the atrophy is distinct the muscles no longer contract to faradism, and, if the motor nerves are tested, they also will be found to have lost irritability. The change depends on degeneration of the nerves, and the muscles usually present the "degenerative reaction" in its characteristic form. The loss of faradic irritability is distinct, in severe cases, as early as the end of the first week, and even by the fifth or sixth day. In a patient, for instance, who is universally, and apparently uniformly, paralysed, one or more groups of muscles may be found to have lost irritability, and in these we know that there will be lasting paralysis and wasting, while the other parts will recover. In a severe case, in which the muscles most affected atrophy completely, the loss of faradic irritability may be permanent. The voltaic irritability remains, increased in degree, for two, three, four, or six months, then slowly falls, as the muscular fibres themselves degenerate, and ultimately, at the end of one or two years, it disappears. It may, however, persist for a long time, if the fibres are stimulated by electricity,—even for three or four years, although the paralysis remains absolute. In rare cases, electrical irritability quickly disappears, even to the voltaic current, from the destructive degeneration of the muscular fibres. More commonly, after six or twelve months, some faradic irritability returns. It may be slight, due to the recovery of a few fibres, insufficient in number to restore any bulk or power, but the new fibres which recover do so perfectly, so that the irritability becomes normal in degree, although the contraction which can be thus produced is very slight in amount. In other cases considerable recovery occurs, so that some power and volume are regained, although the muscles remain below the normal size. In the muscles which are weak but do not waste there is no degenerative reaction, although there may be slight diminution of excitability to each current.

The paralysis, as a rule, is motor only. Sensation is impaired only in extremely rare cases (about 1 in 50), in which inflammation in the lumbar region is so intense as temporarily to impair all the conducting functions of the cord. Thus, in a girl aged five, in whom the permanent state was a symmetrical atrophy of all the muscles below the knees, with a slighter affection of the thigh-muscles and flexors of the hips, the initial general loss of power in the legs was accompanied, for a few days, by complete loss of sensation below the knees. In the similar but more severe case of a boy, aged four, in whom the lasting palsy was general throughout both legs, the anæsthesia continued for two months. When sensation is lost there is always incontinence of urine.

Reflex action is necessarily lost in the parts related to the affected muscles. That from the skin is at first lost where there is weakness, but it returns with or soon after the recovery of power in the less affected parts. Where there is persistent paralysis it remains absent. The myotatic irritability is lost in the same or even greater degree. For instance, no knee-jerk can be obtained if the extensors of the knee are affected even in slight degree. The loss depends on the interruption of the muscle-reflex centre by the disease in the grey matter. In rare cases of severe cervical polio-myelitis, the inflammation spreads beyond the grey matter to the lateral white columns; there is wasting of the arms, but paralysis without wasting in the legs, and in the latter the myotatic irritability may be increased above the normal, so that the foot-clonus may be obtained. In the course of a few months, however, the condition of the legs becomes normal.

A common effect of the disease is to retard the growth of the bones in the affected limb, so that this gradually becomes shorter than its fellow, and the difference in size increases with the amount of general growth. Hence, it is greater, the younger the patient at the onset of the disease. If one part of a limb is more affected than another, the corresponding bone suffers most in growth. The effect is sometimes considerable, when the atrophy is slight. Seeligmüller has described the opposite effect—an actual elongation of the bones, attributed to the fact that the growing epiphyses suffer traction instead of the normal compression.

Those joints that depend for their support on tendons which pass over them become lax when the muscles are paralysed, and the articular surfaces may be no longer kept together. Thus when the deltoid is wasted, the head of the humerus may fall out of the glenoid cavity. The head of the femur may fall from its proper position in the acetabulum, and actual luxation may occur when the paralysis of the muscles is unequal, and the less affected unopposed muscles undergo the secondary contracture immediately to be described. That of the abductors and rotators may cause infra-pubic dislocation, and that of the adductors iliac dislocation.*

During the chronic stage of the disease there is great tendency to the occurrence of permanent shortening of muscles, with consequent displacement of the parts to which they are attached. Grave deformities are thus produced. It occurs only when the several muscles acting on a joint are unequally paralysed; the less affected muscles are no longer subject to the normal extension by their opponents, and hence gradually become contracted, and the contraction is fixed by tissue changes in them, so that they cannot be passively extended. It may occur in muscles which are unaffected, and it is uncertain how far it is favoured by a slight degree of paralysis. Posture also aids the production of these muscular contractions, and so also in the case of the foot does the relative shortening of the limb.

Bedsores are almost unknown, even in the acute stage of the disease. A slight local elevation of temperature in the most affected parts has been observed in the early stage, but subsequently the affected limb is colder than the other. This is due, in part at least, to the want of the aid to the circulation which is, in health, supplied by the muscular action.

The persistent paralysis may occupy the whole or part of one or more limbs. The legs suffer three times as frequently as the arms; the respective percentage being 21 and 68. It is less frequent for both legs or for one arm to suffer, and still rarer combinations are those of both legs and one arm, of both arms, of the arm on one side and the g on the other, while the rarest of all is the affection of leg and arm the same side. The limbs on the left side of the body are more ten permanently affected than those on the right; the difference is eater in the leg than in the arm. The left leg is paralysed alone ice as often as the right.

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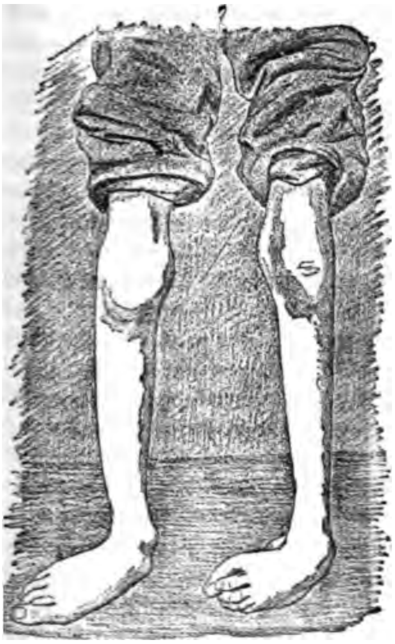
* See Karewski, 'Archiv f. klin. Chir.,' Bd. xxxvii

When the paralysis of a limb is incomplete, the part involved varies uch in different cases, and as different parts of two or more limbs ay be affected, the combinations of palsy which result are extremely ried. The grouping of adjacent muscles is sometimes distinctly that functional association; often it is random; but the most frequent ndition is to have an irregular affection of the muscles that are sociated in the centre. While the grouping is that of function, me of the muscles are only slightly affected, and others suffer with em that are not functionally associated. In the legs, the paralysis, very severe and fortunately very rare cases, may be absolute, volving all the muscles of both limbs. Usually it is partial, and en the muscles below the knee suffer more often than those above the knee. The calf-muscles are affected less frequently or in less degree than the muscles in front of the tibia or the peronei. Hence talipes equinus is a common deformity, and its occurrence is ded by the shortening of the limb, in consequence of which the ot has to be extended to bring the ball to the ground. Either



IG. 108.—Old atrophic spinal paralysis. Talipes valgus in the right leg from paralysis of the anterior tibial; t. varus in the left, from paralysis of the peronei.

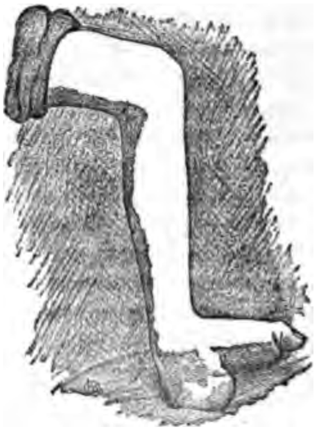


FIG. 109.—Atrophic spinal paralysis, affecting chiefly the calf-muscles, with resulting contraction of the flexors of the foot, causing talipes calcaneus.

the tibialis anticus or the peronei may be more affected, with the result, in the former case, of talipes valgus, in the latter of talipes varus. In the case shown in Fig. 108 the peronei have suffered most in the left leg and least in the right, producing an unsymmetrical but corresponding distortion of the feet. This affords an illustration of another fact, namely, that when there is a partial affection of both legs the paralysis may be unsymmetrical in the two. Much less commonly the calf-muscles suffer more than the others, and there results talipes calcaneus, as in the cases shown in Fig. 109, and the intrinsic muscles of the foot often suffer with those of the leg. In the thigh, the extensors of the knee are affected more frequently than the flexors, and hence flexor contraction is very common, and may even cause subluxation of the joint. The flexors of the hip often suffer, generally with the extensors of the knee. Less commonly the glutei are involved, but never alone.

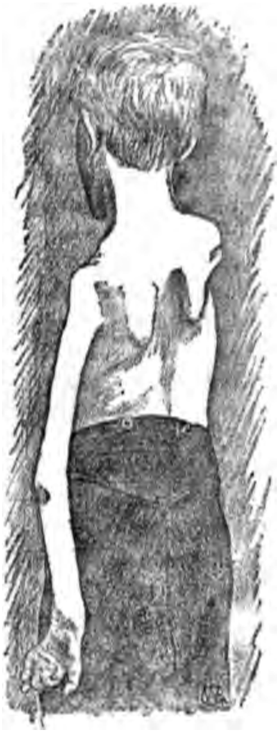


FIG. 110.—Atrophic spinal paralysis, infantile form. Wasting of all the muscles of the left arm from the deltoid downwards, and of the right serratus magnus.

In the arm, almost all the muscles are sometimes affected, as in the case shown in Fig. 110, but all are seldom entirely paralysed. The intrinsic muscles of the hand often suffer: either the thenar muscles or the interossei may be most damaged. An instance of such paralysis of the interossei is shown in Fig. 111. The forearm muscles are frequently affected, but the supinators may escape when the other muscles are involved. The deltoid suffers more frequently than any other single muscle of the arm. It may be paralysed alone or in association with other muscles; occasionally the deltoid, supra- and

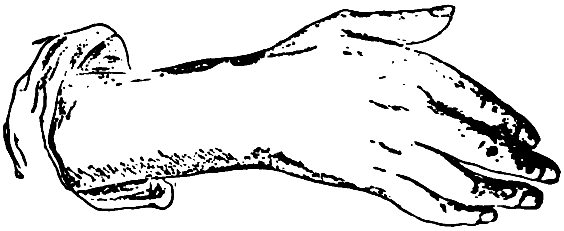


FIG. 111.—Atrophic spinal paralysis (adult form) affecting the interossei and thenar muscles, and also the deltoid.

infra-spinatus, biceps, and supinators are all involved in the "upper arm type" of palsy of Erb* (see p. 91). But other muscles than these may be associated with the deltoid. The irregularity of the grouping is shown by the fact that the triceps is often affected with these muscles. In the case shown in Fig. 111 the deltoid and intrinsic muscles of the hand were wasted, and no other muscles. The serratus magnus is occasionally affected, as in Fig. 110; in this case, although the left arm had suffered severely, as the figure shows, on the right side only the serratus was involved. With the serratus, the upper part of the pectoralis major is sometimes associated in paralysis (as it is in normal function, see p. 30), the lower part being unaffected; it was so in this patient. The middle part of the trapezius and other scapular muscles are occasionally involved. The neck-muscles rarely suffer, but the diaphragm is occasionally paralysed. Although the intercostals and other trunk-muscles are so often weakened in the early stage, considerable permanent atrophy is very rare. Curvature of the spine is sometimes produced in consequence of the patient being allowed to sit up while the muscles are weak. It may be a lateral or an antero-posterior curve, according as the weakness is on one or both sides. I have met with considerable depression of the lower part of the wall of the thorax on the left side from paralysis of the intercostal muscles at the spot, perhaps combined with hindered growth of the ribs. The muscles supplied by cranial nerves are scarcely ever affected. I have once seen complete paralysis of one side of the face, associated with wasting in the limbs, in an otherwise characteristic case, and an instance of affection of the face and tongue has been recorded by Pasteur.†

The *Course* of the disease has been already sketched. There are (1) an initial stage of paralysis, rapidly increasing, often accompanied with fever; the duration of this stage is from a few hours to a week or even more; (2) a stationary period, which lasts for a week to a month; (3) a stage of "regression," during which the palsy passes away, except from certain parts in which wasting occurs; this regressive period usually occupies from one to six months; (4) a chronic stage, during which the atrophy continues; slight improvement may occur, but contractures and deformities are developed. The duration of this stage is indefinite, because, wherever muscular tissue remains and some voluntary power returns, this slowly improves, by increased development of the muscle under the influence of use, and this continues in slight degree for years. This gain is often, however, more than counterbalanced by the interference with growth, and by the occurrence of deformities. Even in the slightest cases complete recovery is extremely rare, and the limb remains smaller than its fellow. On the other hand, death from the disease is equally uncommon, and occurs chiefly in the early stage. It is not improbable

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* See a paper by Dr. Beevor, 'Med.-Chir. Trans.,' 1885.

† 'Lancet,' 1887, ii.

[illegible] children die from the initial disturbance before its nature is recognised. Now and then death occurs at the end of the first week or ten days from universal paralysis or from some such severe [illegible] disturbance as existed in the cases mentioned above, or as followed the spinal lesion in a case in which paralysis in the arms [illegible] the third day of the initial fever and reached its height at the fifth day, faradic irritability being lost by the seventh in many muscles of the left arm; a little rigidity of the neck then occurred, and on the tenth day the patient began to vomit, complained of intense headache and had hallucinations; on the eleventh day he became comatose and died. Bronchial catarrh is an occasional cause of death when the respiratory muscles are weak.

Relapses are very rare, and occur chiefly in the form of a separate [illegible] of the disease. Second attacks are almost unknown; one instance only was met with in the series of 116 cases.

[illegible] beyond those mentioned as part of the symptoms of the disease, are also very rare. As a rule the general and nervous health of the patients, and the duration of life, are uninfluenced by the local malady. In a few cases, however, some other chronic affection of the spinal cord has come on when the subjects of infantile paralysis have reached adult life. Progressive muscular atrophy has been several times observed to start from the paralysed limb, and sometimes to be apparently excited by a fracture of the bone.* Acute and subacute [illegible] in adult life has also been observed, and I have twice seen [illegible] of lateral sclerosis, without atrophy, slowly [illegible] in one case at seventeen, in the other at twenty-eight. In [illegible] the subject of old infantile paralysis there seems [illegible] a disposition, slight though it be, to fresh disease, and [illegible] sclerosis mentioned show that the liability to [illegible] limited, as has been thought, to the grey matter.† The [illegible] the bones has been known to facilitate this fracture; [illegible] bones in the affected limb were broken on three sepa[illegible].

[illegible] For a long time, infantile paralysis was believed to be [illegible] affection, having its seat in the muscles. But improved [illegible] the nerve-centres have shown that changes in [illegible] invariably exist, and have the characters of an acute [illegible] of the grey substance of the anterior horns. The [illegible] between the infantile and adult forms, make it certain [illegible] in most instances, of the same nature. Subacute [illegible] adults has been found, however, to depend on [illegible] some forms that were thought to be spinal, [illegible] certainly identical with the infantile form. [illegible] the changes in the spinal cord in the early stage [illegible] by Dr. D. Drummond, in all probability one of this disease, is by far the earliest on record, and is indeed the earliest possible.[*] A child, five years old, died after a few hours' acute illness. The spinal cord, in the region of the fourth and fifth cervical nerves, presented undue redness of the anterior grey matter. The vessels running from the surface to the cornu were distended with blood. The microscope showed distension of capillaries and minute extravasations in the grey substance, swelling of the neuroglial elements and of the ganglion-cells, which were granular with indistinct processes. Another important early observation is that of Dr. Charlewood Turner,[†] six weeks after the onset (Fig. 112). In this and other somewhat later cases, acute changes have been found in the anterior cornua, more advanced than in Drummond's case. They are often widely spread in slight degree, and attain considerable intensity in one or more foci, usually in the cervical or lumbar enlargements or both. At these spots the anterior horn is softened; sometimes there is hæmorrhagic infiltration, sometimes an actual cavity (Fig. 112, A, B). The microscope shows extravasated blood, often massed along the vessels (Fig. 112, C), and scattered through the grey matter with other cells such as are met with in myelitis. There are also granule corpuscles and other products of degeneration of the nerve-elements. These elements, and especially the motor nerve-cells, have almost entirely disappeared. Where the change is slighter in degree there is a leucocytal infiltration, a few granule corpuscles are seen, while the nerve-cells may be structurally intact, but swollen, and granular. The morbid appearances may be con-

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* [illegible] Méd.' 1880; Dutil, 'Gaz. Méd. de Paris,' 1881.

† [illegible] and Dutil, 'Revue de Médecine,' January, 1884, p. 18.

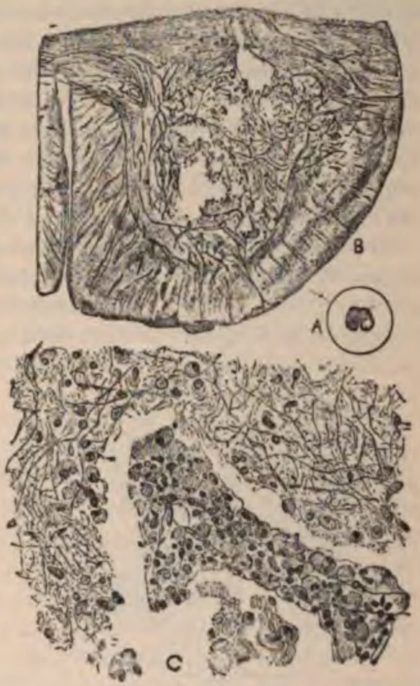

FIG. 112.—Acute anterior polio-myelitis in a child 2½ years old, six weeks after the onset. (After Charlewood Turner.) A. Section through the lowest part of the lumbar enlargement, showing a cavity visible to the naked eye, on the left side. B. Left anterior half of the cord under a low magnifying power, showing destruction of almost the whole anterior cornu. C. Portion of anterior cornu in the upper part of the lumbar enlargement. Numerous corpuscles lie in the granular protoplasm and reticulum of fine fibres. A small artery is encrusted with several layers of corpuscles and nuclei.

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[*] 'Brain,' April, 1885.

[†] 'Path. Trans.,' vol. xxv, p. 203.

fined to the anterior cornua, or may extend in slighter degree into the adjacent lateral column, in which the nerve-fibres near the grey matter may undergo destructive degeneration, and other signs of inflammation may be seen. The posterior columns are always unaffected. In very severe cases there may be signs of slight local meningitis corresponding in position to the region chiefly affected. This region corresponds to the origin of the nerves supplying the muscles in which there is most wasting, and the anterior nerve-roots which arise from this part present the signs of acute degeneration.

Such changes indicate a general or interstitial inflammation of the grey matter. But some observations have revealed only alterations in the motor nerve-cells, which have presented granular degeneration, the basis-substance between them being quite unaffected.[*] This suggests that there is a form of polio-myelitis in which the changes are essentially parenchymatous and not interstitial.

At a later period, years after the onset, the appearances presented accord with those described as commonly found in the early stage, allowance being made for the difference in time. The anterior cornu at one or more places is shrunken (Fig. 113), and in part stains deeply with carmine, in consequence of its nervous structures being replaced by connective tissue, in part less deeply in consequence of "granular disintegration" of the substance. The motor nerve-cells are absent, partially or entirely. Sometimes a few shrivelled cells remain, sometimes one or more groups may be unaffected; or one or two nerve-cells of normal appearance may alone remain (see Fig. 113). Small foci of disease may be seen in the unaffected part of the

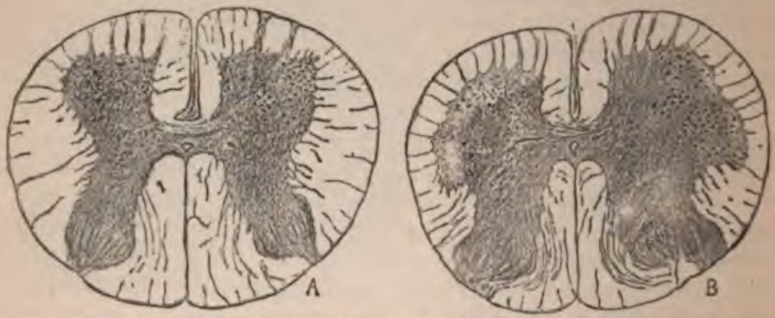

FIG. 113.—Atrophic spinal (infantile) paralysis. A. First lumbar; the left anterior cornu is smaller than the right, and its ganglion-cells have disappeared with the exception of the inner group. B. Fourth lumbar; the whole left half of the cord is smaller than the other, in consequence of the diminution in size of the ant. horn. Of the ganglion-cells only a few remain, belonging to the antero-lateral group. Similar changes existed throughout the lumbar enlargement.[†]

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[*] Rissler, 'Nord. Med. Ark.,' xx.

[†] For these sections I am indebted to Dr. H. Humphreys, of St. Leonards. The paralysis came on at one and a half years of age, two years before death, and involved most of the muscles of the left leg, those of the calf in greatest degree, so that talipes calcaneus had developed.

grey matter, and in these the motor cells may be smaller than normal; the intervening plexus may have perished. When one horn is much damaged, the other may merely present fewer nerve-cells than normal, with perhaps a little condensation of the interstitial tissue. The antero-lateral column is usually lessened in size, and may present slight sclerotic changes, especially in a zone contiguous to the grey substance, whence thickened septa may radiate into the white column. This is evidently due to the extension of an intense inflammation of the grey substance to the adjacent part of the white column. Sometimes a slighter sclerosis can be traced through the lateral column; this is seen chiefly in cases of long duration, and is probably secondary. With extreme damage to both cornua there may be ultimately great wasting of the white columns on each side.* In consequence of these alterations, the affected half of the cord is conspicuously smaller than the other, even to the naked eye, and the difference is greatest where the damage to the grey matter is most intense. The anterior nerve-roots at the most affected part are small and grey, and the degeneration of the motor fibres may be traced down the nerve-trunks. Often a few fibres present a normal appearance although the rest have perished; those that remain may be for the sympathetic system.

The muscular fibres, in early cases, have been found narrower than normal, and in a state of granular degeneration, with an increase in the nuclei of the sheath and of the interstitial tissue. Granules and pigment-masses accumulate between the sarcolemma sheaths. In extreme cases the degeneration proceeds to complete disappearance of the fibres, the place of which is occupied by fibrous tracts, developed partly from the sarcolemma sheaths and partly from the interstitial connective tissue. It is common to find, here and there, a fibre presenting normal appearances. Sometimes fat accumulates in the interstitial tissue, so that the reduction in the bulk of the muscle may be less than that of the fibres. In slighter cases, in which partial recovery takes place, some of the fibres probably regain their normal appearance, even though they have undergone slight granular degeneration. Usually, however, many fibres perish; those which recover are smaller than normal, and the original volume of the muscle is rarely regained. Although there may be a permanent increase in the interstitial tissue, this undergoes contraction and comes to occupy but a small bulk. Muscular fibres are often seen that are larger than normal, and this is not (as has been thought) a transient condition, for it has been observed seventy and forty-five years after the onset.† It is perhaps sometimes a true hypertrophy.‡

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* See 'British Med. Journal,' 1887, p. 187.

† Joffroy and Achard, 'Arch. de Méd. Exp.,' 1889; Déjérine and Huet, 'Arch. de Phys.,' 1888.

‡ Déjérine. It is said to have been met with in muscles totally paralysed; but it is doubtful whether "total palsy" of a muscle excludes voluntary contraction of some fibres in it.

which sensation, as well as motion, is lost. But such cases, which are most rare, differ from true transverse myelitis in the rapidity with which the damage restricts itself to its special seat, and the functions of the white columns are restored. The fact that these are diseased in only a secondary and trifling manner is emphasized by the fact that, even in these cases, there are never the acute bedsores that would surely ensue in a case of true transverse or total myelitis of corresponding seat.

The observations mentioned on p. 366 suggest that there are two varieties of the inflammation of the grey matter, such as we have recognised in myelitis generally; a parenchymatous inflammation consisting in a primary change in the nerve-cells, and an interstitial or general inflammation, as in the common forms of myelitis, involving the nerve-cells only as one of many structures equally damaged by a process that has no special relation to the nerve-elements. The distinction is important, because we should expect to find, in the former cases, a wider slight initial affection than in the others, and finally a more definite relation to function in the parts diseased.

The true pathology of the malady, its relation to its causes, is still obscure. The one salient fact that we can discern, though scarcely define, is the evidence of a blood-state afforded by the general symptoms. That these symptoms are not the consequence of the local inflammation is shown by their disparity in degree and difference in time. It is probable, therefore, that the spinal lesion is not the cause of the constitutional disturbance, but is rather an effect of the cause of the latter. It seems impossible otherwise to understand the extreme variation in the two forms of disturbance. But we have no evidence as to the nature of the blood-state, and there are no other effects, commonly associated with the myelitis, to indicate the general pathological tendency of its cause. The closest analogy is with some forms of multiple neuritis, and this, as we have seen, may co-exist with the spinal lesion, as a consequence, at any rate, of exposure to cold. The variations, alike in the general symptoms, and in the inflammation of the cord, and especially the indications that the latter may be either parenchymatous or interstitial, suggest that the causal state also varies considerably in different cases. We have no indication of the way in which the remarkable relation to season is produced, whether by predisposing the individual or favouring the development of some toxic agent. We must, however, recognise as an essential element the predisposing influence of age, which we may associate with the facts that the structural development of the nervous system is complete, but the function of the nerve-elements must lack the stability that comes only from continued use, and that the period is one of the first serious demand on the functional energy of the grey matter of the cord. We may remember also the vascular activity that all function entails, and the readiness with which the vaso-motor system

The bones have been found, not only shorter than normal, but also smaller, with their angles rounded, the compact substance more uniform, and the Haversian canals smaller (Joffroy and Achard).

The brain has been almost always found normal. In one case with extensive paralysis, of long duration, some atrophy was found in the motor region of the cortex (Sanders). It is probable that some acute changes would be found in cases that die with the cerebral symptoms already described.

No observations have as yet been made on the acute primary polyneuritis that seems occasionally to coincide with the inflammation of the cord. It is probable that, in the cases in which the nerves are tender in the middle of their course, the sheath will be found inflamed, and only the motor fibres will present such signs of secondary degeneration as always result from the lesion of the cord.

PATHOLOGY.—Both the character of the lesion and its mode of onset suggest that it is inflammatory in the majority of cases. In rare instances, as already stated, the almost instantaneous onset makes it probable that the lesion is different in character, is vascular in its nature, probably hæmorrhage. These cases have, moreover, the features of a primary hæmorrhage, not of hæmorrhagic myelitis. Signs of inflammation do not precede the sudden onset. Thus these cases, while they belong to the clinical group of acute atrophic paralyses, cannot be placed in the pathological category of poliomyelitis. They usually differ from the inflammatory cases in another respect, that the lesion is confined to one spot in the cord, whereas in the cases that are certainly myelitic it is frequent for several parts of the cord to be damaged.

The symptoms are well explained by the pathological facts that have been ascertained, extended by the simple inferences that are indicated by the symptoms. The lesion interrupts the motor path, causing immediate palsy, which results from damage to the cells and nerve-plexus of the grey matter, whether slight or severe. The recovery of the slightly damaged structures restores the voluntary path, and even if the fibres from the cells degenerate, they seem to regain normal nutrition by regeneration when the cells recover. But when the motor cells are destroyed the fibres from these degenerate permanently. As the damage is usually far wider than the destruction, the initial palsy is more extensive than the persistent atrophy, and the extent of such is purely a matter of this relative area of trifling and severe degree of lesion. Nerve-cells that are destroyed are never renewed, and hence the permanence of the muscular wasting that depends on such destruction. When a few nerve-cells escape destruction, a few nerve-fibres recover and a little muscle-tissue remains, too slight, it may be, to be of use. The occasional interference with the conducting tracts in the white columns is explained by the obvious extension of the inflammation, which, when very intense, may even spread through all the structures of the cord, as in the cases in which sensation, as well as motion, is lost. But such cases, which are most rare, differ from true transverse myelitis in the rapidity with which the damage restricts itself to its special seat, and the functions of the white columns are restored. The fact that these are diseased in only a secondary and trifling manner is emphasized by the fact that, even in these cases, there are never the acute bedsores that would surely ensue in a case of true transverse or total myelitis of corresponding seat.

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DIAGNOSIS.—This rarely presents any difficulty except in the early stage. When the initial paralysis is passing away, and the wasting is distinct, the nature of the case is sufficiently evident, and is corroborated by the electrical reactions, by the loss of reflex action, and by the absence of any impairment of sensibility. At the onset, however, the symptoms may readily be misinterpreted. The most common error is to attach too much importance to vomiting, and to regard the attack as simply gastric. General disturbance is naturally ascribed to some general cause until nervous symptoms manifest themselves. Even then there is risk of error. On the one hand, as we have seen, a diagnosis of a general disease is not always relinquished when paralysis appears, but the latter is regarded as secondary. On the other hand, especially in young children, the existence of paralysis is often overlooked at first, and it is supposed that the child does not move because it is prostrate. This error will not be made if it is remembered that mere prostration never causes total immobility, and *à fortiori* it does not produce local immobility. When the pyrexia ceases, and the loss of power persists and increases, the existence of paralysis is always unmistakable. The initial general disturbance must then be regarded as part of the disease unless there is the clearest evidence of its independent nature.

In adults, the danger of mistaking paralysis for prostration is considerably less, but, on the other hand, the general symptoms are as likely to be misinterpreted as in the case of children. The rheumatoid pains, which are so common, are usually regarded as evidence of acute rheumatism, especially when the affection follows exposure to cold. Whenever rheumatoid pains are not localised in the joints, and especially when they are spontaneous, and not influenced by movement, the possibility of their spinal (or nerve) origin should be remembered, and other indications of spinal mischief, such as local loss of power, tingling or formication, should be carefully watched for and receive due weight.

Of other spinal cord diseases, all chronic lesions are distinguished at once by the onset. From other acute diseases, moreover, the distinction is only difficult in the early stage. As soon as the initial palsy begins to lessen, and the muscles in one part lose faradic irritability and begin to waste, the nature of the case admits of no doubt. The diagnosis may be thus made with certainty after the end of the first week by the electrical reaction. If we find loss of faradic irritability, it is certain that the characteristic alteration in voltaic irritability will follow. The isolated induced shocks should be used, instead of the induced "current," because any harmful stimulation of the sensory nerves is thereby avoided.

Acute transverse myelitis is only simulated when the inflammation of the grey matter is bilateral and so intense as to extend to the white columns, but the age of the patient generally suggests correctly the nature of the disease. The myelitis of childhood is polio-myelitis. The pseudo-transverse variety—polio-myelitis, transverse through its intensity—is seated in one of the enlargements; the true transverse form usually occurs in the dorsal region. Real difficulty is confined to adults, in whom there is extensive cervical or lumbar myelitis involving the grey matter. In young adults, polio-myelitis is more likely; this is indicated also by what is best described as a regressive onset in contrast to one that is progressive. In the former, a wide extent is quickly reached and the symptoms then tend to lessen; in the latter, the morbid process is a longer time reaching its height: in the former, the symptoms clearly show that the most severe affection is of the anterior grey matter; in the latter, severe anæsthesia or trophic disturbance may point to an equally intense lesion of the other parts of the cord. If attention is paid to the character and significance of the symptoms, the difficulties will soon vanish; and the cases that present them are really few. Most perplexities will, indeed, be prevented by the recollection of the fact just mentioned, that primary myelitis in children is always polio-myelitis. The separation of subacute from acute myelitis of the grey substance is, to a large extent, arbitrary. The symptoms are similar, but the onset is less rapid and occupies more than a week. The distinction of diphtheritic paralysis and of multiple neuritis is from the subacute rather than the acute spinal atrophies, and is considered in the next section.

The diagnosis from paralysis of cerebral origin is usually easy. In cerebral palsy there is never loss of faradic irritability or extreme muscular wasting, or loss of the muscle-reflex action. In the spinal affection there is no trace of the mobile spasm that is common in infantile hemiplegia. Any cerebral symptoms which may attend the onset of polio-myelitis are subordinate in significance to the state of the muscles, as evidence of the permanent lesion. Convulsions at the onset of infantile spinal paralysis are general; those that result from a cerebral lesion are usually unilateral or commence locally.

The exceedingly slow onset of pseudo-hypertrophic paralysis, developing gradually, as it does, with the child's growth, ought to render its confusion with polio-myelitis impossible. A reasonable doubt can only arise in a slight case in which the onset was unnoticed or forgotten. I have once known slight atrophic palsy of the extensors of the knee to induce the habit of putting the hand on the knee in rising from the ground—an action often thought to be peculiar to the pseudo-hypertrophic disease, but acquired when weakness of the extensors occurs in early childhood from any cause. If this is not known, it may puzzle the observer.

Diseases outside the nervous system which have been mistaken for infantile paralysis are chiefly those in which local pain interferes with the movement of the limbs, and the child is young. I have known, for instance, the mistake to be made in the case of hip-joint disease, necrosis of the femur, and the affection termed "scorbutic rickets," in which there is enlargement of the shafts of the long bones, extreme pain on movement, and spongy gums. In all these cases, a careful examination will show that movement is interfered with only by the pain; there is no actual paralysis, and there is no interference with reflex action. The preservation of the knee-jerk is often of great significance; it at once excludes atrophic palsy in any case in which the extensors of the knee are weak.

The points above described will always enable the diagnosis to be made. It is impracticable to enumerate the distinctions from every disease with which confusion is possible, for experience shows that there is no form of palsy with which a common disease, such as this, is not at some time confounded.

PROGNOSIS.—The danger to life is probably greatest when there is severe constitutional disturbance, and is in consequence of this, often before the development of the characteristic paralytic symptoms. In the stage of paralysis, there is peril only when the chief disease is in the cervical region, and respiration is interfered with; but this danger is small unless an attack of bronchial catarrh is intensified by the palsy. Definite cerebral symptoms involve danger proportioned to their character and degree, but it is generally less in reality than in appearance. In the vast majority of cases the disease involves no immediate danger to life. But children are left with little power of resistance to other morbid influences, and occasionally succumb to some other illness, as an acute specific disease, or an attack of bronchitis, a few weeks or months after the onset of the paralysis.

As soon as the paralysis has become stationary, *i. e.* has not increased for twenty-four hours, the danger of further extension is small. But the question at once arises, and is anxiously asked,—What will be the permanent condition? Will there be lasting paralysis? An answer cannot be given until the end of the first week or ten days, and then only by means of an electrical examination. Whatever muscles, at the end of that time, have lost faradic irritability, will certainly waste, will remain for a long time paralysed, and will probably be permanently affected in some degree, slight or severe. On the other hand, if there is no loss of irritability at the end of ten days, but it is apparent at the end of a fortnight or three weeks, the wasting will be slighter in degree, and some ultimate recovery may be anticipated even in the most affected part. Where there is no loss of irritability, the paralysis will pass away in the course of a few weeks, or at most of a few months. Where faradic irritability is lost early and completely, the wasting will be rapid and great, and it is unlikely that there will be much recovery of power or nutrition, although some use may be regained. The return of faradic irritability that has been lost is a favorable indication; it signifies nerve-regeneration, and will be followed by an increase in voluntary power. Without an electrical examination it is necessary to wait longer before a prognosis can be given, until distinct wasting on the one hand, and improvement on the other, indicate the regions in which the paralysis will persist and in which it will pass away. Even then the prognosis must be much less definite.

In the chronic stage, the prospect of ultimate recovery depends on the rate at which wasting developed, on the electrical reaction, and on the duration of the case. Where there is no sign of returning power at the end of three months, very little recovery will occur; the nerve-cells are destroyed and their renewal is impossible. The preservation of voltaic irritability (of the muscular fibres) is so far satisfactory that it shows there has been no destructive degeneration of the muscles; and if voluntary power is increasing, it indicates favorable conditions for its exertion, but it does not lessen the grave significance of persistent palsy and the absence of faradic irritability that indicates persistent nerve-degeneration. On the other hand, if, at the end of one or two months, some faradic irritability can be still detected, although low in degree (*i. e.* elicited only by a strong current), improvement is probable, and may be considerable in the course of a few months. It is necessary, in the case of children, to remember, and to warn the friends, that the growth of the most affected limb will be hindered, and that this, in the case of the leg, may render the effect of the paralysis more obtrusive by its interference with the gait. Otherwise they are distressed by what seems to them an increase in the disease, but is really compatible with continued improvement.

TREATMENT.—The treatment of the acute stage of the disease is essentially the same as that of myelitis, already described, and the rules and principles already stated need not be here repeated. In the initial stage it should be that of the general state, guided by any special causal indications that may be detected; such as free sweating, followed by salicylate of soda or salicin, in a case distinctly due to exposure to cold. We may hope that future observations will afford us some indications regarding the means of counteracting other blood-states that apparently exist at the onset, and are concerned in the production of the lesion. At present we are without this help, and can only treat the initial stage as we should any other local inflammation, directing our treatment to the symptoms that may be present, and the conditions that apparently underlie them. The child should be kept at perfect rest, on the side, and warmth applied over the affected part of the cord by poultices or fomentations. The marked relief these give when there is spinal pain, make it probable that they exert a beneficial influence in all cases.

In such a disease—in which there is a natural tendency for the morbid process to cease to spread, and then to lessen in extent—the difficulty of ascertaining the effect of treatment on the lesion of the spinal cord is very great. Full doses of belladonna or ergot have been credited with the control and arrest of the morbid process, but only on the evidence of a coincidence in isolated cases, the value of which is small in a disease so irregular in its course. Either may apparently be employed, however, without fear of harm.

When the acute onset is over and the spinal lesion has become stationary the careful management of the early stage must be continued for a time, because a relapse or recurrence, however rare, is not entirely unknown. Such care is especially needed in cases in which the constitutional disturbance has been prolonged, or has continued after the onset of the spinal symptoms, or the latter have come on in distinct stages. Under these circumstances, perfect rest should be maintained for one or two weeks more. The same prolonged care is needed when there is persistent tenderness in the limbs or any indications of an independent neuritis. Where wasting is taking place, some tenderness of the nerves and of the muscles must be expected to accompany the process, and this, being purely secondary in nature, does not call for special treatment.

When the acute stage is over, and the condition of the patient is stationary or improving, tonics are generally both useful and needed, especially iron and quinine. Strychnia may be given in all cases, but it should be commenced only two to four weeks after the disease has become stationary. Although clear proof of its utility is not forthcoming, and cannot indeed be expected in a disease in which merely damaged nerve-elements always tend to recover, the drug has a certain influence on the nutrition of the structures that are specially affected, and it is reasonable to suppose that it promotes their recovery, and is capable of rendering this more perfect. But an agent that has so powerful an influence on function (and therefore on nutrition) is likely to do harm, rather than good, until normal conditions are being restored. If function cannot respond properly to a stimulus, it is not likely that nutrition can be influenced aright, or that its derangement can be other than increased. Hence, the more severe the lesion the longer time should elapse before strychnia is commenced, and the smaller the dose that should at first be given. It is probably never either necessary or desirable to give it by hypodermic injection in this disease.

The disease frequently occurs at the age at which any illness causes the hindrance to general development that constitutes the condition known as rickets, and this fact should be remembered in treatment. In the case of children who are still in the period of the first dentition, it is, therefore, wise to anticipate the danger by giving, during convalescence, those agents that have most influence in counteracting the tendency, especially cod-liver oil and iron; the lacto-phosphate of lime and iron is especially useful.

The use of electricity is an important part of the treatment, although for reasons very different from those that first led to its use, and it is desirable that they should be clearly comprehended to secure its benefit and prevent its harmful or useless employment. There is no evidence that it can or does influence the process of recovery of the damaged elements in either the spinal cord or the nerves. Moreover, as long as the nutrition of these is actively disordered, its stimulating influence is likely to increase the derangement rather than promote the restoration of a normal state. The reasons for its use depend on the fact that the disease entails nerve-degeneration, and are essentially the same as those that justify and determine its employment in other nerve-lesions, and have been already explained. The muscular fibres whose nerves are degenerated suffer changes in nutrition, and ultimately perish, if no nerve-regeneration occurs. While the influence normally exerted by and through the nerve-fibres is in abeyance, the muscles are destitute of this influence, and without any functional stimulation. If they are excited, from time to time, by electricity, their sensitiveness to stimulation is distinctly increased, and this not only to electricity but to the voluntary stimulus. This is clearly shown by cases that have been untreated; the muscles may not respond at all to the first application, but when they have been galvanised two or three times a distinct contraction may be obtained, and, within a week, some voluntary power may return. Wherever cell and fibre have perished, nothing that electricity effects can be of service; but where there has been damage, not destruction, and the fibres slowly recover, but regain their influence on nutrition, and their capacity for conveying impulses, only after some months, the failure of muscular nutrition may be disproportionately great, and may even render useless some regained nerve-power. This result electricity is probably able to prevent. It does not, as far as we can perceive, prevent or even lessen the visible wasting of the muscles; it is powerless to counteract the effects of a destructive lesion, but it does seem to hasten recovery from the effects of partial damage and to lessen the ultimate degree of this. It does so solely by its influence on the muscular tissue, and only voltaic electricity can stimulate this tissue when the nerve-fibres are degenerated. The mode of application should be the same as for nerve-lesions. The muscle is only stimulated when the circuit is interrupted, as by repeatedly stroking the muscles with one terminal (a sponge, or conductor covered with water-holding material, well wetted with salt and water), which should be lifted from the skin between each stroke. The other terminal is kept still; it may be placed on the upper part of the muscle, where its nerve enters it. By some, this terminal is placed on the spine, over the affected region (and then a larger flat terminal is employed), under the impression that the electricity may influence the morbid process in the spinal cord; but there is no evidence even that the spinal cord is reached by electricity so applied. When applied to the muscles, the strokes may be made with the negative terminal, since each pole will influence the tissue, and the normally greater irritability to the negative often persists. The application can be made perfectly well by an intelligent nurse, since the diffusion of the voltaic current is so great that it is sure to reach the affected muscles. Each time the sponge is placed on the skin, the affected muscles should be seen to move or swell up in slight contraction, and it is desirable to use as many cells of the battery as will produce this result. Sometimes this cannot be done without causing so much pain as to distress the child, but in many instances the distress is really due to a needlessly strong and painful current having at some time been used, and an amount of alarm produced that the child never gets over. It is on this account that it is so important to make a test examination with great care, and to employ the faradic shock rather than the current. Emotional disturbance may generally be avoided by commencing with a very weak current. It is better, indeed, to commence with no current at all, applying the sponges in the way directed, so that the child may be familar with them, and cease to fear them. Then two or three cells may be used and the strength gradually increased day by day. In this way a current strong enough to cause contraction will often be tolerated. If, however, in spite of these precautions, this strength cannot be employed without distressing and disturbing the child, the attempt to obtain muscular contraction should not be persevered in, but only such a current employed as does not disturb the child. An influence on nutrition, if slighter, is still exerted, and if the application is continued for a longer time it is probable that equal good is done. The electrical treatment may be commenced at the end of the third or fourth week after the onset. It should not be used earlier, lest it excite increased disturbance in the spinal cord. The application need only be made to those muscles in which faradic irritability is lowered or lost. Other muscles will either recover perfectly without its aid, or (as in the case of paralysis of the legs from implication of the lateral columns in the cervical region) will be uninfluenced by electricity.

Another measure which should be employed is systematic rubbing of the limbs. This stimulates the circulation, which is always defective, as the blue, cold surface shows. It no doubt also increases the movement of the fluids in the tissues outside the vessels, and so probably increases the interchange of material, and promotes nutrition. The muscles should be daily rubbed and gently kneaded, upward rubbing being especially useful. No liniment is required; the uncovered hand answers best. Cod-liver oil is sometimes rubbed on; some of it is probably rubbed in, without good or harm. Great care should be taken, at all times, to keep the affected limbs as warm as possible.

In all cases in which the trunk-muscles are weak, even in slight degree, bronchial catarrh must be guarded against with great care. In such cases the muscles of respiration are weakened, and although normal breathing may not be impaired, the diminution in strength may render an acute bronchial catarrh rapidly fatal, and this even months after the onset of the paralysis.

When some voluntary power is regained, the systematic use of the muscles is of great service, and should be carefully attended to. Systematic planned movements may generally be adopted, and an improvement of muscular nutrition and power may be secured much earlier than if the limbs are left to the chance influence of such movements as a child spontaneously adopts. Most of the good that is done by "movement cures" may be obtained by such simple muscular exercises as can be readily arranged with the help of a little consideration on the part of the doctor, and can be efficiently carried on at home, given the necessary perseverance on the part of those who have charge of the patient, and which, unfortunately, is less often forthcoming. Such movements need to be specially arranged to meet the exigencies of each case, so as to call into action those muscles that are weak but not powerless, or that may supplement the muscles that are paralysed.

A very important element in the management of the chronic stage is the prevention and treatment of the muscular contractions, and of the deformities to which these give rise. The contractions cannot be entirely hindered, but they may often be prevented reaching a high degree by careful attention to the position of the limbs, and by watching for and treating the earliest indication of shortening of the muscles. It is needless to discuss in detail the varied influence of posture in these cases; careful and frequent examination of the patient will show whether any deviation from normal relations is being developed, and its counteraction is chiefly a matter of common sense and a little practical ingenuity. Of especial importance, however, are the prevention of the curvatures of the spine which are caused by allowing the patient to sit up before the back-muscles have regained the necessary power, and the prevention of the shortening of the flexors of the hip and knee-joints that occurs when the patient is allowed to lie in bed with the legs drawn up. More difficult of prevention is the contraction of the calf-muscles, often aggravated in consequence of the lessened growth of the limb. Something may be done during the process of rubbing, for the prevention of deformities. While the contracted muscles are rubbed upwards, they should be extended gently but firmly. Thus, if the calf-muscles are shortened, the foot should be steadily pressed upwards while the calf is rubbed. A slipper with elastic straps to a knee-band, may be worn at night, often with great benefit. For developed deformities, due to great shortening of the muscles, surgical treatment by splints or tenotomy is usually necessary, and is often important because it enables the patient to use the limb in ways that would be impossible without it.

For this and other reasons, mechanical appliances are of unquestionable value. Counteracting deformities, and supplementing weak muscles, they often enable a child to walk, who could not do so

without their aid, and power is often increased to a remarkable exte
in indirect ways. In all these cases, however, the instruments requi
frequent attention and alteration to adapt them to the changes due
growth, and patience is required in the management of these cas
through their long and tedious course.

Two facts deserve a final emphasis. At the end of six months
possible recovery is nearly completed, and certainly at the end of
year the lesion has become a cicatrix, and further improvement will
merely by the slow growth of the muscle that has recovered, und
the stimulus of use. Secondly, this process of slow improvement
the result of use will go on for years,—will go on whatever treatme
is adopted, and whether special measures are employed or not. T
therapeutic specialist who carries on his treatment month after mon
will claim it as his achievement, but it would occur equally witho
his aid. From its nature, however, as already stated, it may pr
bably be always augmented by some contrivance to increase the i
fluences that are really improving the power of the muscles.

SUBACUTE AND CHRONIC ATROPHIC SPINAL PARALYSIS (SUBACUTE AND CHRONIC POLIO-MYELITIS).

Under this designation cases have been described of a miscellaneo
character, in which paralysis, followed by muscular atrophy, comes
less rapidly than in the acute form, its development occupying fro
ten to thirty days, in the cases which are termed "subacute,"
from one to six months or even more, in those which are ca
"chronic." Many cases included under this name differ muc
their characters, and are sometimes described as "chronic myel
but among the cases placed, even until lately, in this group, are
of multiple neuritis. Most subacute atrophic palsies are due to
disease; almost all, indeed, which present symmetrical palsy.

The cases of spinal atrophic paralysis, in which the onset
acute, may be placed in three classes.

(1) Cases of subacute polio-myelitis which differ from th
form, already described, only in their less rapid developme
present the same initial general disturbance and wi
and the same recovery, except in a limited region in
wasting occurs. They are thus distinguished fr
also by the irregular distribution of the sympto
have been adults. Little is known of the e
further than that it certainly sometim
and is probably sometimes the resu
symptoms and general history
difference from the acute fo

(2) Cases are met with t
the fact that their course is
chronic, occupying from

arrest followed by improvement, the more or less rapid onset is followed by slower gradual increase, until at last wide-spread chronic muscular atrophy is developed. Many of these are really cases of progressive muscular atrophy with a subacute onset. For example, I have more than once known a case of this disease, ultimately typical, to begin by a subacute atrophic paralysis of the extensors of the wrists. These cases will be considered in the account of that disease. Others present differences from that malady and symptoms of more irregular character, which show that the lesions are distributed through various structures in the cord, and these must be regarded as cases of chronic myelitis involving the anterior cornua. This form also is chiefly confined to adults. It occasionally results from cold, and sometimes from injury, as a fall on the back. Intemperance and venereal excesses are also said to cause it. The wide-spread muscular atrophy which sometimes results from lead-poisoning is probably of this nature. The muscles at first present the degenerative reaction, or normal faradic irritability with increase to voltaism; sometimes, however, there is a loss to both faradism and voltaism. The sphincters usually escape. In many cases, the affection exhibits a persistently progressive character; muscular atrophy slowly increases and extends, until the case ultimately resembles one of progressive muscular atrophy, and the patient dies at the end of one or two years from exhaustion, or from interference with the respiratory movements. In other cases, the atrophy, after slowly progressing for many months, becomes stationary, and considerable improvement may ultimately take place. This course is seen especially in traumatic cases. Few observations on the pathological anatomy of the affection have been made. Cornil and Lépine found, in one case, at the end of four years, softening of the lower part of the spinal cord, chiefly in the anterior cornua, disappearance of the ganglion-cells, increase of the connective tissue, and sclerosis of the white substance around the anterior horns.

The diagnosis from the first form rests on the slow onset and progressive course of the disease. Although the degenerative electrical reaction may be found, it is frequently absent, and this is another distinction from the acute variety. On the other hand, the fact that paralysis precedes wasting is a distinction from progressive muscular atrophy. In the most chronic form, however, this distinction fails, and cases are met with which present a gradation between the two diseases.

The prognosis is grave except in traumatic cases, but is influenced by the observed rate of progress, and the presence or absence of any indications of arrest. When the result of injury, considerable improvement is not uncommon, and it is remarkable how great a degree of paralysis and atrophy may, in these cases, ultimately pass away almost completely. The treatment of the disease must be influenced by the rapidity of its onset. In the subacute stage and form it should be conducted on the same principles as that of acute cornual myelitis. In the chronic form the treatment must be the same as for progressive muscular atrophy.

(3) Many subacute and chronic cases, which have been described as atrophic spinal paralysis, are peripheral, not central in their nature, and are cases of multiple neuritis, the symptoms and diagnostic indications of which have been already described. The history of the subacute spinal disease has been largely written from cases of multiple neuritis. In the statements just made, this fact has been kept in view. While multiple neuritis may simulate closely chronic poliomyelitis, it is certain that the converse is also true, and the central affection has been sometimes thought to be peripheral. The facts at present available suggest that the central and peripheral structures of common function possess common susceptibilities to the action of morbid influences, and there is still danger that the two classes may be confused. The most important criterion is the greater irregularity in distribution of the central disease,—the more perfect symmetry of the peripheral affection. The presence of tenderness of the nerve-trunks, and of the structures to which the nerves are distributed, is of more value, when it exists, than is the suggestion afforded by its absence that the disease is central, because, in the periphery, only fibres of one function may suffer; we may then have none of the symptoms that we commonly associate with neuritis, and look for as proof of its existence. In purely motor neuritis there may be no tenderness or pain. But such an affection is either unilateral or perfectly symmetrical. Imperfect symmetry indicates an affection of the cord.

ACUTE ASCENDING PARALYSIS.

Ascending paralysis, which commences in the legs and ascends to the muscles of the trunk, the arms, muscles of the neck, the diaphragm and the pharynx, is a feature of several diseases—meningeal hæmorrhage, ascending myelitis, &c.; but it occurs also in cases in which the spinal cord, after death, appears healthy to both naked eye and microscopical examination. Such cases have, however, certain characteristic features which make it desirable to distinguish the condition from other diseases, and the affection has, therefore, been provisionally termed "*acute ascending paralysis*," in the absence of any evidence of its nature. It was first described by Landry in 1859, and hence is often called *Landry's paralysis*. Acute ascending paralysis is a most formidable malady, most cases proving fatal in a few days. Its nature is mysterious, but recent discoveries regarding multiple neuritis have disclosed instructive analogies between the two affections. These have, indeed, led some observers to the opinion that this ascending paralysis, without organic central lesion, is an affection of the nerves. As we shall see, it is not probable that this opinion is correct, although it is possible that the nerves, as well as the centres, are sometimes affected.

CAUSES.—The etiology of the disease resembles, in general, that of acute multiple neuritis more nearly than that of any other affection. The disease affects males more frequently than females. It occurs chiefly between twenty and forty years of age, but has been observed, in rare cases, in older and younger persons, and even in children. Some sufferers have been the subjects of alcoholism. Severe exposure to cold has been the apparent cause in many instances. In other cases the disease has occurred under conditions such as cause toxæmic states, or after the occurrence of some known blood-disease. Thus it has occurred during convalescence from some general disease, smallpox, diphtheria, typhoid fever. It has also followed febrile diseases of obscure nature. In these cases, it should be noted, the disease has followed at an interval of a week or a few weeks. It also sometimes succeeds some traumatic process, such as a wound, and here also after an interval, and usually when the wound was apparently healed. I have known it thus to follow an attack of pelvic cellulitis. In these respects it closely resembles polyneuritis. The disease has been repeatedly observed in the subjects of syphilis, and has apparently been arrested by the treatment for this disease.* The circumstance that when some malady or wound preceded the disease, the former was often of a trivial character, prevents surprise at the fact that, in many cases, no influence could be traced to which the disease could be ascribed.

SYMPTOMS.—Premonitory symptoms have been noted in some cases —general malaise, pains in the head and back, tingling in the extremities—for a few days or a week before the onset. The first definite symptom is usually weakness of the legs, often commencing in one and spreading to the other. The weakness increases rapidly, so that the power of standing is lost, sometimes in a few hours, sometimes at the end of two or three days; it goes on to complete paralysis, with relaxation of the muscles. As the legs become motionless, the muscles of the trunk become weak, first of the pelvis, loins, and abdomen, then of the thorax. The weakness next invades the arms; either the upper arm muscles or those of the forearm and hand may be first attacked, and one arm is often weakened before the other. The paralysis of the arms may become absolute, like that of the legs, or some power of movement may remain. The diaphragm and neck

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* Much weight should not be placed on this fact as proof of causal relationship, because (apart from the fallacy of independent cessation) it is probable that the treatment that is effective against the virus of syphilis is also effective against other blood-states that depend on agents of similar nature.

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[illegible], brief pyrexia attended the onset, and in a few others moderate [illegible] disturbance (2° or 3°) occurred at a late period. Profuse sweating has been occasionally noted. Enlargement of the spleen [illegible] noted by Westphal), such as occurs in other acute diseases, is [illegible] frequent.

Thus the chief feature of the disease is an almost purely motor paralysis, progressive in character, with relaxation of the muscles during the acute stage of the disease. It usually has an ascending course, but irregular cases are met with in which the order of invasion [illegible] varied. Thus the arms are occasionally involved before the legs, [illegible] diaphragm before the intercostals; the bulbar symptoms have [illegible] known to precede the others, and I have seen them reach a high degree with a fatal involvement of the cardiac centre, before the upper muscles of respiration were affected or the hands quite powerless. Analogous cases have been recorded by others, the special features [illegible] course being those characteristic of the disease. In some cases, however, of irregular course, changes have been found in the electric [illegible] of the muscles; it is probable that, in such cases, the peripheral nerves are affected, although we cannot, on this account, at present separate these from the typical cases of the disorder—a point [illegible] be explained in connection with its pathology.

[illegible] disease varies considerably in the rate of its progress. It may [illegible] course and end fatally in forty-eight hours. Death usually [illegible] either respiratory or cardiac paralysis—the latter in the irregular course. A large proportion of the fatal cases last [illegible] week. On the other hand, the disease may only attain its [illegible] the end of two, three, or even four weeks. In some cases, [illegible] of the same character, the paralysis of the limbs has not complete. There may be a rapid extension of the paralysis to a [illegible], and it may then cease to spread, and the limbs last and [illegible]ted may slowly regain power. A patient may lie for days [illegible] condition of universal palsy, save for a little respiratory power, [illegible] improvement begin. As a rule, the order of recovery of the [illegible] is the opposite to that of their invasion. In favorable cases recovery of power is usually slow; two or three months often [illegible] before the weakness entirely disappears. Occasionally there [illegible] a more rapid improvement, and the patient has been well in [illegible] weeks.

PATHOLOGY.—As a rule the most careful and skilled examination has failed to discover any morbid appearance in the spinal cord, [illegible] or muscles. Minute hæmorrhages, met with occasionally, have probably occurred during the last moments of life. In a few cases, apparently similar to the others, slight scattered indications of inflammation have been found in the grey matter of the cord, with degenerative changes in the ganglion-cells (Immermann); but, as a [illegible] the central nervous system shows no trace of lesion. Of late more attention has been paid to the peripheral nerves, and slight indications of acute changes, both parenchymatous and interstitial,[*] have been found in them, although in some cases they have been apparently normal. In addition to the enlargement of the spleen observed during life and found also after death, the mesenteric glands have been found swollen, and also the closed follicles of the intestines. Organisms were found in the glands by Baumgarten, but most observers have searched for them without success in all cases of typical character.

These negative facts, taken in conjunction with the conditions under which the disease occurs, and with the course of the malady, have suggested the idea of a toxic influence acting on the nerve-centres; and this idea receives support from the discovery that acute swelling of the spleen is common, and of the lymphatic glands not rare—lesions that indicate a morbid blood-state. The limitation by function of many toxæmic palsies, *e.g.* that of accommodation, affords confirmation of this view, since the isolated acute paralysis of a functional centre, not anatomically separate from others, is known only as a consequence of a toxic influence. Further confirmation is afforded by the fact that cases of acute multiple neuritis, certainly due to a toxæmia, may run a similar course, and that indications of such commencing neuritis may actually be found in this disease. But this does not exclude a central process. It has, indeed, been conjectured that the nerves are always the structures impaired, but this view is rendered unlikely by the common absence of altered excitability of the muscles, since the motor nerves seem never to escape invariably in peripheral neuritis of any variety, however common their freedom from affection in the type. At the same time this does not exclude the periphery: the nerves may suffer also in some cases, and there may, indeed, be gradations to the cases of acute multiple neuritis of ascending course described at p. 128. If we ask on what nerve-structures this toxic influence is usually exerted, we meet with the difficulty that while it is clearly upon some part of the motor path, the integrity of the muscles shows that the interruption is not in their nerves or nerve-cells, and their relaxation, with the prolonged impairment of myotatic irritability, suggests that the structures interfered with are not far from the motor centres. Curara abolishes the function of the termination of the lower segment of the motor path, *i. e.* of the nerve-endings in the muscles. It may be that, in this disease, some toxic influence impairs in an analogous manner the function of the termination of the upper segment, paralyses the ramification in which, as we have seen (p. 175), the pyramidal fibres must end in the grey matter, and by which they are connected with the lower segment. It has been already pointed out (p. 213) that the nutritional stability of the termination is probably lower than that of any other part of each segment, since it is the part furthest from the nerve-cell from which the fibre proceeds, and on which its nutrition depends. The greater length of the fibres for the lumbar region may render their termination more susceptible than that of the fibres for the arm, and hence the ascending course of the paralysis. It is, moreover, readily conceivable that the effect extends in slight degree to other structures in the grey matter—those, for instance, which intervene between the sensory and motor cells; and thus the loss of reflex action and myotatic irritability is explained, as well as the muscular relaxation, since, as we have seen, muscular tone must be ascribed to a reflex process. The structures affected may vary somewhat in different cases, and thus the state of reflex action may vary. The facts of diphtheritic paralysis also show that the effects of such a toxic influence may be revealed, in varying degree, by visible changes having the characters of inflammation, and we can thus understand that indications of such inflammation in the grey matter may be occasionally met with. The bulbar symptoms come under the same explanation. The motor path through the nuclei of the medulla has the same pathological relations as that which passes through the grey matter of the spinal cord. The separate affection of nerve-structures, distinct from others only in function, is common in the phenomena of toxic actions. There are no facts to show the nature of the blood-state, except those that suggest its alliance with the influences that act on the peripheral nerves; multiple neuritis should be studied in connection with this disease. These facts suggest that the virus is a product of the preceding growth of organisms, at least in some cases; and if so, it is natural that the organisms themselves should not be found.

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[*] Eisenlohr, 'Deut. med. Wochenschr.,' 1890, No. 38.

Diagnosis.—The disease is recognised by the rapid development of paralysis, usually ascending, with relaxation of the muscles, and with loss of reflex action, without considerable pain or loss of sensation, and if the patient survives, without wasting of the muscles or change in electrical irritability. The latter characteristic distinguishes the disease from acute atrophic paralysis, while the absence of pain in the back and of spasm is a distinction from meningeal hæmorrhage. The diagnosis from general ascending myelitis has been already considered; it rests especially on the involvement of all the functions of the cord in inflammation. The distinction from multiple neuritis has been mentioned in the account of that disease (p. 140), but, it may be, is not always absolute, since the nerves probably suffer, as well as the cord, in some cases of ascending paralysis. The distinction from polyneuritis, founded on the course of the ascending form, is subject, moreover, to the reservation that while ascension to the arm through the trunk is a feature of the central disease, this is not excluded by a more irregular course, and the character of the individual symptoms must be taken into consideration (see also p. 383).

Prognosis.—The affection is one of extreme gravity. The danger to life is in proportion to the interference with respiration and with the functions of the medulla oblongata, especially with the cardiac centre, and also to the rapidity with which the palsy comes on. But the cases which develop with comparative slowness are not devoid of danger. Even when the symptoms only reach their height at the end of three or four weeks, death may occur in the same way as in the more rapid cases. On the other hand, recovery has been known although the patient has lost all power of motion at the end of the second day. The danger is great as long as the symptoms are increasing, and only when distinct improvement can be recognised is it justifiable to anticipate recovery. The earlier the bulbar symptoms appear, the more serious is their significance. Mental symptoms also increase the gravity of the prognosis, as they usually indicate a severe blood-change, the effects of which on the nervous system are likely to reach a high degree.

TREATMENT.—During the early stage of an attack of acute ascending paralysis, in the absence of other indications, the treatment should be that suitable for myelitis, since, at the onset, the diagnosis between the two diseases can never be certain. A warm bath, or still better, a vapour bath, should be given if the symptoms followed exposure to cold. It should be followed by counter-irritation over the spine by a long, narrow mustard plaster. More energetic counter-irritation, even the actual cautery, has been recommended. The body should be kept in as perfect rest as possible. In a very few cases have drugs appeared to exert any influence on the course of the disease, and the malady is so rare that experience accumulates slowly. Salicylate of soda seems to deserve a trial in cases that follow exposure to cold. Ergotin has been given, and one case in which it was used deserves special mention. The patient was a man aged fifty-seven, who, a week after exposure to cold and wet, complained of a feeling of weight and weakness in the legs; the temperature rose to 103°; the loss of power gradually became complete in the legs and spread to the arms, without loss of sensation. At the end of the second day there was difficulty in swallowing, in articulation, and in breathing, and death seemed near. Ergotin was given every hour, and during the night the patient took twenty grains. In the morning the bulbar symptoms were better, the arms stronger, and there was a trace of motor power in the legs. The patient rapidly improved, and at the end of a week was well. If swallowing becomes difficult, care must be taken to administer a sufficient amount of nourishment, either by the rectum or by the nasal tube. Several cases have been recorded in persons who had had syphilis, in whom arrest of the disease followed the administration of iodide of mercury. The chief lesson of modern researches is certainly that we must look for the means of effective treatment to the neutralisation of the toxic influence on which the malady apparently depends. At present, however, nothing has been ascertained regarding antidotal agents available in the acutely active stage of these toxæmic states. It is, however, probable that mercury is capable of doing this in some cases, and that when the causation is obscure, and the disease develops slowly enough to permit mercurial treatment, it is wise to adopt this irrespective of the fact of preceding syphilis. In cases that follow traumatic lesions, especially if there have been any indications of septicæmia, full doses of perchloride of iron offer, I believe, the best means of destroying the activity of the blood-state. Urgent symptoms require the same treatment as in myelitis.

PARALYSIS FROM LESSENED ATMOSPHERIC PRESSURE; DIVERS' PARALYSIS.

Divers, and especially those who work in caissons, at such a depth beneath water that they are exposed to considerable pressure, may become paralysed soon after their return to the surface.[*] Apoplectic attacks and hemiplegia also sometimes occur, but paralysis of the legs is by far the most common effect, and it is, therefore, clear that the spinal cord suffers in greater degree than any other part of the nervous system. Miners have been said to suffer also, but this is doubtful, because a pressure equal to at least an additional atmosphere seems necessary for the production of the symptoms. Most of the subjects of the disease have worked at a depth of from forty to ninety feet below the surface of water, and under a pressure of two to four atmospheres. In the extensive works involved in laying the foundations of bridges, in which many men have been employed, a considerable proportion of the workers have suffered in some measure; but severe degrees of affection are rare, because the conditions under which they occur can be avoided. They only occur in those who have been exposed for more than a certain time, which is the shorter the greater the pressure. At a depth of ninety feet beneath the surface, immunity from severe symptoms (and commonly from all symptoms) is obtained by reducing the periods of work to an hour. The danger is greater in those who have had several previous periods of work on the same day, and especially if slight symptoms have been experienced after one of these. It is also greater in those unaccustomed to the conditions. I have met with one case of hemiplegia in a diver after only half an hour's work in a well at ninety-six feet, but the man had been down twice before with only intervals of rest of half an hour, and the attack passed off in the course of an hour. In another case paraplegia occurred after working for an hour under only fifty feet of water, but the man had worked on the preceding day at a depth of one hundred and twenty feet below the surface. It is evident, therefore, that the power of resisting the dangerous influence varies, and that predisposition to suffer may be caused by the conditions which also induce an attack.

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[*] See, on this subject, Babington and Cuthbert, 'Dubl. Quart. Journ.,' 1863, p. 312 (cases at Londonderry); Eads, 'Med. Times and Gaz.,' 1871, p. 291 (cases at St. Louis); Leyden, 'Arch. f. Psychiatrie,' ix, Heft 2; and Moxon, 'Lancet,' 1881, ii, 529.

It is not during exposure to the abnormal conditions, but on returning to the normal atmospheric pressure, that the symptoms come on; often immediately, and always within half an hour or at most an hour of the return to the surface. The onset is usually preceded by other symptoms, and especially by pains in the ears and in the joints. The latter is very common, and occurs after a much slighter degree of exposure than is needed to produce paralysis. They are felt chiefly in the larger joints, and may continue for days and even longer. Very rarely there is also swelling of the joints.

It is important to note the conditions under which the symptoms come on. Not only do those who are unused to such work seem especially liable to suffer, but there are also considerable individual variations in the degree of liability.

Paraplegia or hemiplegia may occur, but the former is far more common. The onset of the paralysis is sudden. The legs feel heavy and are found to be weak, and in a few minutes the patient is unable to move them. Sensation is often lost as well as motion, and in all severe cases the sphincters are affected. The arms are seldom involved. In slight cases the loss of power is incomplete, and one leg may be more affected than the other. The impairment of sensation is often imperfect or irregular. If the paralysis is incomplete the power may return in the course of a few days, but in severe cases the palsy usually lasts for weeks or even months, and it may be permanent. Death may occur at the end of a week or more, in the same way and from the same causes as in acute myelitis. When the paralysis is hemiplegic it is generally moderate in severity and transient, passing off in a few hours or days. It may be even still more brief, as in the case referred to above, in which a diver, after half an hour's work ninety-six feet below the surface, suddenly felt tingling across his loins, and that his right arm and leg were almost powerless; in half an hour he had recovered sufficiently to walk home. If cerebral symptoms are more intense, they are generally very severe, with sudden loss of consciousness, continuing as deep coma, irregular breathing, and indications of cardiac paralysis. Such cases usually end in death in the course of a few hours.

Pathology.—The most probable explanation of these cases ascribes them to the escape from the blood of gases, with which it has become charged during the exposure to the high pressure. It is certain that a great excess of gas must be dissolved in the blood during the exposure, and that the amount, at least of oxygen and of carbonic acid, contained in the blood when the person emerges from the caisson must be vastly greater than normal. It must, moreover, be greater the longer has been the exposure, since the absorption of the excess will be a gradual process, taking place through the lungs in respiration, but it will be more rapid as the pressure is greater. These conditions agree with observed facts. After returning to the normal atmospheric pressure, the excess of gas probably passes off gradually by the lungs in most instances; but if extreme, gas may escape from the blood within the body. The occurrence of this has been proved experimentally (Hoppe-Seyler and P. Bert). Gas, so escaping, is no doubt quickly reabsorbed, but must exert a pressure capable of arresting the function of the structures of the nervous system. If abundant, it may conceivably rupture these. Further, the special effect on the nerve-centres may be connected with the position of these within cavities that are practically closed. These conditions, coupled with the extremely circuitous course of the blood from the cord, may explain the incidence of the effects on the nervous system.

The few ascertained facts harmonise with this pathology. Such escape of gas has been proved to result from diminution of the atmospheric pressure.* In many cases no visible lesions have been found, and it is obvious that gas, widely effused, may exert dangerous pressure, and yet may be speedily removed, so that no indication may remain of the cause that has completely arrested function.

It was once thought that the symptoms were due to local hæmorrhages, but extravasations are seldom met with, even in the results of experiment, and it is certain that they take but a trifling part in the production of symptoms. The only positive pathological observation agrees with the opinions above stated. Leyden† found (in a case of characteristic paraplegia) small irregular fissures in the mid-dorsal region, chiefly within the posterior and hinder parts of the lateral column. The fissures were filled with round-cells, but contained no red blood-corpuscles, and from their well-defined edges they were certainly not produced by the infiltration of the cells found within them. The only explanation that is satisfactory, or in any harmony with their features, is that they were produced by the sudden escape of gas, and were afterwards occupied by the round-cells. The physical firmness of the cord is far less in the dorsal region than in the enlargements; and probably, as Leyden suggests, this is the reason why the dorsal region suffers structural damage from the escape of gas more than other parts. It is evident, however, that such escape may occur, in the brain, for instance, even to a fatal degree, and leave no traces unless it occurs so rapidly as to cause laceration of the tissue. The absence of hæmorrhage of the ordinary character may be further explained by the fact that the effused air will resist the effusion of blood. Some other anomalous features, such as the transient character of the symptoms in some cases, are also explained by the peculiar nature of the lesion, which may exert a wide-spread and considerable

* Hoppe-Seyler, 'Müller's Archiv,' 1887; P. Bert, 'Comptes Rend.,' 1871–2.

† 'Arch. f. Psych.,' ix, Heft 2.

influence, and in a short time may have vanished by the reabsorption of the compressing air. Hæmorrhage must be regarded as an accidental consequence, and even the production of such fissures as were found by Leyden may not be a necessary part of the lesion, even in grave cases. The mechanical effects of the gas may be considerable, and arrest the function of the nerve-elements, without the passage of the gas outside the capillary vessels.

The fact that the spinal cord suffers more than other organs may be partly due to the plexuses through which the blood can return only slowly to the lungs, where its relief from the surcharge of gas is effected. The same considerations apply to the brain. In both organs the escape of air is more instantly disastrous than elsewhere, and, indeed, may not only occur, but act in a peculiar way, on account of the position of the organs within chambers that are, to a large degree, closed. The conditions are too complex to permit us to follow them in detail, or analyse fully the mechanisms that determine the effect on the nerve-centres. The escape of the excess of gas in the lungs can only take place gradually, as successive quantities of the surcharged blood pass through the capillaries of the air-cells. If, in consequence of the slowness of the circulation in the cord, any escape of air occurs in its capillaries, the effect of this will be still further to hinder the local circulation, and to favour the further escape of gas. In this way we are able to discern something of the mechanism which determines the special affection of the spinal cord. The less the external support the less will be the resistance to the escape of gas, and the incidence of the lesion on the posterior part of the cord may be in part at least determined by the adoption of the recumbent posture when the first symptoms are perceived.

At the onset, when the first symptoms are experienced, it is probable that a return to a greater degree of pressure might arrest the development of the mischief. It is certain that, in all cases of exposure to a high degree of pressure, the transition to the normal pressure should be made gradually. When developed symptoms indicate that a definite lesion of the cord has occurred, the further treatment must be conducted on the same principles as in acute myelitis—the morbid process that has, in fact, been set up.

HÆMORRHAGE INTO THE SPINAL CORD; HÆMATO-MYELIA.

Primary hæmorrhage into the spinal cord, sufficient to cause symptoms, is a very rare disease, and it is even more rare than is suggested by the cases now and then recorded as such. We have already seen that hæmorrhage may accompany myelitis; a considerable extravation may occur when the inflammation is only commencing, during the state of congestion, and while the symptoms are slight. Such cases are easily mistaken for primary hæmorrhage. The risk of error is not always removed by pathological examination. Inflammation results from hæmorrhage, and when indications of myelitis are found about a clot, it may be impossible to say whether these are primary or secondary. It is probable that many cases of secondary myelitic hæmorrhage have been regarded and described as primary, and it is possible that a few cases of primary hæmorrhage have been regarded as secondary. One writer, Hayem,[*] goes so far as to deny the occurrence of primary non-traumatic hæmorrhage; but such an exclusive view is unwarranted. It is certain, however, that the history of primary hæmorrhage has been largely written from uncertain data, and will need extensive revision when a sufficient number of exact observations have accumulated.

Etiology.—The rarity of hæmorrhage into the cord is especially great in comparison with the frequency of hæmorrhage into the brain. The difference probably depends on the tortuous and long course of the arterial path to the cord, whereby the vessels are preserved from the high pressure which is the chief cause of the degeneration, dilatation, and rupture of the cerebral arteries. Miliary aneurisms are not found within the spinal cord. Hæmorrhage is far more common in males than in females. It may occur at any age, and has been met with in young children, even so early as seven months,[†] while some of the subjects have been in advanced life; but it is most common between twenty and forty, *i. e.* during the first half of adult life. Many cases occur in young adults apart from injury or obvious exciting cause, and apart also from initial myelitis, which accounts, however, for some of the cases. It has been met with as a consequence of a hæmorrhagic tendency, as in a case in which it succeeded severe epistaxis, to which the patient, a young man of twenty-four, was liable.[‡]

Of immediate causes, injury is the most frequent, especially falls which involve a severe concussion of the spine; the spinal column may or may not be injured at the same time. Over-exertion and exposure to cold have in rare cases preceded the onset. Chronic alcoholism and sexual excess have been thought to predispose. In one case within my knowledge, an extensive hæmorrhage into the grey substance at the top of the lumbar enlargement resulted from coitus four times repeated, the symptoms commencing suddenly during the fourth act. Minute extravasations are often found after death from diseases which interfere with respiration and cause venous congestion, and they are especially frequent in maladies which, at the same time, cause functional excitement of the cord, as tetanus and all severe convulsions. They cause no symptoms, and are probably produced during the last moments of life. They have been termed "*accessory*." The diseases of the cord that lead to *secondary* hæmorrhage are chiefly inflammation, tumours, and cavities in the cord. The last-named condition is especially important. It is probable that hæmorrhage occurs more readily in spinal cords that contain a congenital cavity or fissure, due to an arrest of development, and surrounded by embryonal neuroglial tissue. Hence this condition will be found in disproportionate frequency in cases of hæmorrhage.

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[*] 'Des Hémorrhagies intra-rachidiennes,' Paris, 1872.

[†] Clifford Allbutt, 'Lancet,' 1870, vol. ii, p. 84. Numerous hæmorrhages were observed in the grey matter of the lumbar and cervical enlargements (probably polio-myelitic, but occurring the day after a fall) in a child of four by Chaffey ('Path. Trans.,' 1885).

[‡] Sinclair, 'Lancet,' 1885, ii, p. 1043.

Pathological Anatomy.—The minute extravasations just mentioned, as met with after asphyxial and convulsive diseases, are found in both the grey and white substance, but especially in the former. They are usually microscopic, or visible to the naked eye as minute red points, distinguishable from distended vessels only by their slighter resistance to a stream of water. The extravasation may occupy the perivascular sheath, or the cavity in which the vessel lies, or extend between the nerve-elements. The larger non-traumatic hæmorrhages, which cause symptoms, always begin in the grey substance, and are often confined to it, extending into the white columns only when large in size. The vessels of the grey substance are more numerous than those of the white, have less external support, and probably undergo more considerable changes in state. The effusion forms a cavity in the cord, sometimes rounded, sometimes irregular, in transverse section, and half an inch or more in vertical extent. The cord is enlarged at the seat of the hæmorrhage, and this may be visible externally as a dark swelling the size of a nut or a bean. Very rarely the hæmorrhage tears the layer of cord which limits it, and blood, usually only in small quantity, escapes into the membranes. The tissue adjacent to the clot is usually broken down, stained, and softened, and inflammatory changes may be recognised in it with the microscope, just as in the neighbourhood of hæmorrhages into the brain. As in the latter, the effused blood slowly undergoes changes in tint, becoming first rusty and then yellow, and ultimately a cyst may remain. Several extravasations may coexist, usually in the same part of the cord. In contrast to this *focal hæmorrhage* there may be an infiltration of the grey matter with punctiform extravasations, which may appear, until closely examined, to be a single hæmorrhage. The tissue between these small extravasations is broken down. It is probable that this form is always secondary to myelitis, as in the case mentioned in the foot-note on p. 391. In such secondary cases a careful microscopical examination usually reveals indications of inflammation much more extensive than the area affected by the hæmorrhage. A growth into which hæmorrhage occurs is usually a glioma, unaffected parts of which will be found in the neighbourhood of the extravasation. The cavities in the cord into which blood may escape are sometimes of considerable vertical extent. I have known a fissure in the posterior column to be filled with blood through almost the whole length of the cord. This condition thus favours both the occurrence and the extension of hæmorrhage.

SYMPTOMS.—Slight symptoms, "prodromata," have been observed in some cases, chiefly in the form of trifling sensory disturbance, tingling, &c., in the limbs afterwards paralysed. They have existed for a few hours or days, or even for two or three weeks before the onset. It is probable, however, that these have been cases of secondary myelitic hæmorrhage, and that there are no premonitory symptoms in primary extravasations. The actual onset is always sudden; the symptoms attain a considerable degree in the course of a few minutes, but they sometimes increase during one or two hours, when the hæmorrhage is from a small vessel and slowly increases in size—probably augmented by the rupture of other vessels which are torn in the tissue lacerated by the blood. Occasionally there has been transient loss of consciousness without any cerebral lesion, probably from the upward influence of the shock. Rarely the onset has been by a series of sudden augmentations of the symptoms. Sometimes the symptoms come on during sleep. The suddenness of the onset is the characteristic of the disease.

The symptoms which thus develop vary according to the seat and extent of the extravasation. In the majority of cases there is paraplegia, complete motor and sensory paralysis up to the level of the lesion, with loss of power over the sphincters. Pain commonly but not invariably accompanies the sudden palsy; it may be felt in the spine or in the sacrum, round the trunk, at the front of the thorax, or at the epigastrium, sometimes seeming to pass thence through the trunk to the spine, or it may be felt in the legs. If in the spine it is local, and does not extend through a considerable length of the spine, as in meningeal hæmorrhage, and there is not the initial spasm and rigidity which characterise the latter disease. This pain may precede the palsy, even for half an hour or an hour; it begins suddenly, and is no doubt due to the first slight extravasation, which afterwards increases (when some resistance yields), and effects the compression that produces the paralysis. Thus a girl aged fifteen was seized with sudden sharp pain, referred to the sternum at the mammary level, and to the corresponding region of the spine; this continued for half an hour, when the legs suddenly became powerless and insensitive. It is highly probable that, when there is such initial medial or bilateral pain, the hæmorrhage occurs in the central region of the grey matter, perhaps from one of the commissural or anastomotic arteries (see p. 188), and that the pain is due to the irritation of the fibres of the posterior commissure, in which the paths for pain cross the middle line. The spinal column may be tender opposite the affected spot. Usually the paralysed muscles are relaxed. Sometimes they are the seat of early clonic contractions, or these may come on a few days after the onset. When the hæmorrhage is in the cervical region all the limbs are powerless; one arm is often affected before the other. The state of reflex action varies according to the seat of the disease; if it is at first abolished it quickly returns in the legs (unless the hæmorrhage is in the lumbar enlargement), and it soon becomes excessive unless destroyed by secondary myelitis—a not unfrequent consequence. In one case there was a remarkable initial increase of myotatic irritability a few hours after the onset, probably irritative, and quickly giving place to loss.[*] Vaso-motor and trophic changes in the skin are common and often intense; there is often vascular dilatation; bedsores readily form, and cystitis may result. Frequently the secretion of sweat is increased for a time. The temperature is normal at the onset, but it generally rises in the course of a few days from secondary inflammation in the cord.

The palsy developed at the onset usually continues for a week or ten days, although the pain may lessen. The symptoms do not always increase during the stage of inflammation, perhaps because this only involves the structures which are already impaired by pressure; but if the patient has been brought near to death by the primary hæmorrhage, the secondary inflammation may end life. Occasionally, moreover, symptoms of an ascending or descending myelitis may come on, and the former may cause death by its interference with the muscles of respiration. This extension is greatest probably in cases of myelitic hæmorrhage, in which the extravasation is merely an incident in the course of a commencing inflammation, but it seems also to occur in cases of primary hæmorrhage. The slow extension upwards of the symptoms, during the first week, may be very distinct; and the occurrence of descending inflammation may be shown by the loss of reflex action and by the failure of electric irritability in the muscles, sometimes only in those supplied from the upper part of the lumbar enlargement.

The symptoms pass into a chronic stage, improvement being usually slow. Some lasting loss of power remains in most cases, and there is often some permanent muscular wasting on account of the frequency with which the hæmorrhage is in the cervical or lumbar grey matter. Occasionally there is rapid recovery up to a certain point; a hæmorrhage of small size may abolish conduction in the white columns by the suddenness with which it compresses them, and the effects of the pressure may quickly pass away, while those remain that are due to the destruction of the grey matter.

Diagnosis.—The diagnosis rests on the actually sudden onset of the symptoms, and on the occurrence of pain, in a limited region, as part of these. The former is evidence of a vascular lesion (rupture or closure), and the latter shows an acute irritation of the nerve elements, such as may be produced by their laceration, but not by mere deprivation of blood. It must be remembered, however, that we cannot assume that symptoms which come on during the night's sleep are of sudden onset. Neglect of this consideration sometimes causes a mistake in diagnosis. The mode of onset is a sufficient distinction from all other organic diseases, except hæmorrhagic myelitis and meningeal hæmorrhage. The former (really a variety of hæmorrhage) is distinguished by the existence of slight symptoms before the sudden attack. We are not justified in regarding as primary hæmorrhage any case in which premonitory symptoms existed for more than a few minutes, unless such symptoms were so pronounced and sudden in onset that they might have been due to a definite extravasation, afterwards increasing. Initial fever (within the first six hours) always makes myelitis probable, provided there is no other cause for it. The distinction from meningeal hæmorrhage has been mentioned in the account of the symptoms, and in the description of that disease.

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[*] Sinclair, loc. cit.

Prognosis.—In all cases in which the symptoms are considerable in degree or wide in range, the danger to life is great, and remains great until they begin to subside. The prognosis is better when the disease is in the dorsal region than when it is in the enlargements, for the same causes as influence the prognosis in myelitis. It is better when sensation returns in the course of a few days, but if the enlargements are affected other sources of danger remain considerable. Early trophic changes also render the prognosis worse, for they show an intensity of degree that may involve grave danger. After the onset is over, the forecast must be guided by the general principles that determine the prognosis in acute myelitis.

Treatment.—The treatment of hæmorrhage into the substance of the cord is the same as that of hæmorrhage into the membranes (p. 295). The measures requisite are few, simple, and all-important. Absolute rest and the prone position are to be secured before anything else is thought of. Ice should be applied to the spine over the seat of the hæmorrhage. The bowels should be opened freely, and full doses of ergot or ergotin may be given: ʒss of the liquid extract or five grains of ergotin may be given by the mouth, or three grains of ergotin may be injected under the skin, and the dose may be repeated two or three times, at intervals of two hours. A few large doses are probably more effectual than smaller doses continued for a longer time, since the hæmorrhage probably does not go on for long. The after-treatment must be that for myelitis. The disease is one of those in which most of the good that can be done by treatment—and in few diseases is the opportunity more urgent—rests with those in whose hands the patient is immediately after the onset.

DEGENERATIONS OF THE SPINAL CORD.

A large and important class of diseases of the spinal cord consists of those in which there is a slow degeneration of the nerve-elements, with an overgrowth of connective tissue, and in which structures are affected that have a common function, while others that have a different function escape even when they are adjacent to the elements that are diseased. Affecting thus functional "systems," they are termed "system diseases." This term has been used in several senses, more or less special (even based on the developmental relations of the structures), and hence the question whether a given malady is a "system disease" or not, is one to which various answers have been given. The term is here used in its widest and simplest sense, as meaning an affection of the structures that have a function either the same, or so far allied that they work together as parts of one system, distinguishable as such from other sets of structures. A "system disease" may involve all the structures of the system, or only some or one of them; its chief distinction is from a random disease that involves structures irrespective of function—merely, for instance, because they are contiguous, or supplied by the same artery.

In "system diseases," the primary change is, as a rule, in the nerve-elements, and the overgrowth of interstitial tissue is secondary. The process is analogous to that which occurs in the secondary degeneration of nerve-fibres, in which the first change is certainly in the nerve-elements, the destruction of which is followed by an overgrowth of nuclei and supporting tissue, amounting ultimately to a "sclerosis," as it is termed. The process is by some regarded as a chronic parenchymatous inflammation, an inflammation beginning in the proper functional elements of the organ, but it seems undesirable thus to widen and loosen our conception of inflammation; so far as the process is concerned the question is one of name rather than of nature.

Another question of much greater interest is the relation of the two elements in the process, the wasting and the growth, the atrophy of the nerve-tissue, the hypertrophy of the connective tissue. The failure of nutrition in the one causes an increased energy of nutrition in the other. We have seen this relation in the nerve-fibres. The degeneration of a fibre is attended by an active growth of its nuclei and protoplasm. The nutrition of the two elements, the neural and the adventitial, is evidently connected in the closest manner, but in inverse course. It is important to recognise the fact that the process of growth of the connective-tissue elements is an active manifestation of nutritional energy, which, when once excited, may be to some extent independent of its cause. It may, when very active, pass beyond its proportional limits, and be greater than corresponds to the nerve atrophy that causes it; invading adjacent structures as if an independent process. It may, when very rapid, have some of the characters of an interstitial inflammation, and even an acute inflammation. Such excess is quite unusual; as a rule the secondary process is subordinate and proportioned to the primary change.

These degenerative diseases fall into certain types so far as the spinal cord is concerned, but they are subject to some variety of combination even there, and to great diversity of association with degenerative changes elsewhere. The affection of the cord is of either the sensory or the motor elements, or both. The type of the former is tabes; of the latter, either spastic paraplegia or muscular atrophy, according as the lower segment at the motor path is affected or free. Other combinations of affection will be better understood when the diseases are described.

The degenerative diseases are not numerous, but they are of great importance. They are degeneration of the anterior cornua and anterior root-fibres, causing muscular atrophy; degeneration of the posterior columns and posterior root-fibres, causing locomotor ataxy; and degeneration of the pyramidal tracts, causing spastic paraplegia, a disease the exact pathological position of which is not quite certain. These degenerations may be variously combined, and some combinations need separate description.

LOCOMOTOR ATAXY; TABES DORSALIS.

The malady thus named is the most common chronic disease of the spinal cord. It consists in a degeneration in the posterior columns of the spinal cord, or the peripheral sensory nerves, or both, and is manifested, when considerable, by inco-ordination of movement, peculiar pains, defective sensibility, and loss of the myotatic irritability (muscle-reflex action), of which the knee-jerk is the most convenient manifestation. When slight in degree, the symptoms may be limited to the pains and the loss of the knee-jerk. The name "locomotor ataxy" was given to the disease by Duchenne; "tabes dorsalis," or "wasting of the back," is a term applied by Hippocrates to certain symptoms supposed to be due to venereal excess, and long ago limited in Germany to symptoms believed to depend on atrophy of the spinal cord. It then included all chronic paraplegias, but was further restricted to this disease by Romberg. It has lately obtained wider use on account of the discovery that inco-ordination may be absent when the disease is slight in degree.

The symptoms vary much in different cases. Besides the varieties thus produced, there are two allied affections that ought not to be classed with it. One is the so-called "hereditary ataxy." The other is that in which both weakness and inco-ordination co-exist from the first; "ataxic paraplegia" it may be called. Each differs from other varieties with sufficient constancy to merit separate consideration. These varieties are not included in the following account.

HISTORY.—The inco-ordination of movement and other symptoms of the disease were frequently noted, during the first third of this century, in cases of disease of the spinal cord, but such cases were not distinguished from those with actual loss of power. The inco-ordination was found to be associated with disease of the posterior columns by Stanley.* The first really exact account of the disease was published in 1847 by Todd,† who distinguished the cases with inco-ordination and without weakness, from simple paraplegia, and, apparently not aware of Stanley's observation, he inferred (from the character of the symptoms and his theory that the posterior columns contain fibres connecting segments of the cord at different levels) that the posterior columns would be found diseased; and he verified this inference by finding in two cases disease of these columns. The credit of the discovery of the disease belongs, if to anyone, unquestionably to Todd, and few diseases can with greater truth be said to have been "discovered." Four years later (in 1851) Romberg described the disease and the lesion in the posterior columns, but he failed to exclude loss of power from the symptoms. Russell Reynolds, in 1855, gave an accurate description of the symptoms, and, in attributing the ataxy to muscular anæsthesia, was the first to give what we must now regard as the true explanation of the chief symptom.‡ A series of cases was described by Gull in 1856 and 1857. Türck first observed with the microscope the wasting of the fibres in the posterior columns. Duchenne in 1858-9 published an independent and very able analysis of the symptoms of the disease, and gave it the name "locomotor ataxy." He obtained for it (with the help of Trousseau's "Lectures") the recognition that previous descriptions had failed to secure, and achieved such a degree of success that the malady is called in France "Duchenne's disease." If any name is attached to it, that of Todd alone can be right.

CAUSES.—The disease is much more frequent, in this country at least, in urban than rural populations. Inherited influence is to be traced only in a small proportion of the cases, perhaps in not more than 10 per cent. It is usually a general neurotic heredity, manifested by such diseases as insanity, epilepsy, and other degenerative diseases f the nervous system. Instances of this are—father epileptic; ather insane; two sisters insane. Direct inheritance of the disease s extremely rare (the special hereditary form being always excluded). A boy with distinct symptoms and optic nerve atrophy was the son of a man who presented characteristic indications of the early stage of abes (Remak). But in most instances of this character the inheritance is effected by means of syphilis.

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* 'Med. Gazette,' [illegible], and 'Med.-Chir. Trans.' vol. xxiii.

† 'Cyclopædia of Anatomy and Physiology,' vol. iii, p. 721.

‡ Reynolds, 'Diagnosis of Diseases of the Brain, &c.,' 1855, "Anæsthesia Muscularis." Certain words are worth quoting:—"It appears most probable that [illegible] [illegible] of [illegible] [illegible], and that the locality of lesion is [illegible]" (p. 106).

Males suffer far more frequently than females, the proportion being about ten to one, and this implies some proclivity inherent in the male sex. A like preponderance of males obtains in a disease that has some alliances with tabes—general paralysis of the insane. The middle period of adult life is that in which locomotor ataxy usually commences. No less than half the cases begin between thirty and forty, one quarter between forty and fifty, and rather less than a quarter between twenty and thirty. It rarely begins after fifty, but I have once known it to develop at sixty-six. Under twenty it is still more rare, but cases are met with as early as ten, and even in quite young children, the subjects of the cause next described.

Among the individual causes, one overshadows all the rest—the influence of syphilis. A very large proportion of the sufferers have had, at some previous time, constitutional syphilis, either distinct secondary symptoms or an indurated sore. The proportion is almost as large in the upper and middle classes as in the lower. Of fifty consecutive cases of the disease in men, seen in private consulting practice, no less than twenty-nine, or 58 per cent., gave a history of a chancre known to be hard, or of secondary symptoms; and eight others had had a venereal sore of unknown nature. In the lower classes the proportion is higher, and has been estimated at 80 per cent., or even more. The ascertainable facts are certainly below the real facts, as I have pointed out elsewhere.* When a deduction is made for possible accidental coincidence, there remains at least one half of the cases in which numerical coïncidence must depend on causal relationship. It is probable, indeed, that, taking all cases, a causal proportion of three quarters would be nearer the truth. In women also antecedent syphilis can often be traced, although rather less frequently than in men. At the same time the facts are even more difficult to ascertain, since in married women syphilis so often runs a latent course. But in the cases of tabes that succeed syphilis the lesion is not syphilitic in histological character; it is as degenerative as in the cases in which syphilis can be excluded, and it is not influenced by the treatment for syphilis. Hence it must be regarded as a degenerative sequel of syphilis rather than as a true syphilitic disease. It is probably the influence of syphilis that determines the greater incidence on the urban than on the rural population, and the preponderance of cases in middle life. Inherited syphilis is also capable of causing the disease, and is to be traced in all cases in children. After the acquired disease the interval between primary syphilis and the first symptoms of tabes varies from one to twenty years. It is, however, rarely less than three years, and in most cases it is between six and twelve years. In rare cases the first symptoms occur during the active stage of syphilis.*

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* 'Lettsomian Lectures,' 1889.

It is certain, however, that syphilis is not the only cause of the disease. In a few cases, less than 10 per cent. of the whole, it can be excluded with confidence. The causes operative in these can be traced also in some of the patients who present a history of syphilis, and in such cases the causation of the malady is probably complex.

One of these causes, which can sometimes be clearly traced, is injury, such as involves concussion of the spine. The immediate results of the injury, whether slight or grave, transient or lasting, are followed by the symptoms of the degenerative malady. In one case a man fell from a height on to the deck of a ship; transient paraplegia resulted, but six weeks after the accident the man presented extreme ataxy, with good power, and no knee-jerk (Arnold). I have known the symptoms to develop gradually a few months after a fall from a horse. Exposure to cold and wet has occasionally preceded the onset so directly that it must be regarded as a cause. In other cases the disease has succeeded excessive fatigue and over-exertion, and also certain acute diseases, especially (it is said) acute rheumatism and typhoid fever; it follows diphtheria in rare cases, but the ataxic form of diphtheritic paralysis, passing away, is far more common than true tabes. (See also section on Pathology.) Alcoholic excess has been noted in some instances, and the influence of this cause is intelligible since it is known to cause sometimes such a form of peripheral neuritis as occurs in tabes. Indeed, it is strange that this cause cannot be traced more frequently. Sexual excess has been supposed, by some, to be a cause; but its influence can rarely be detected, and its significance is uncertain, since sexual excitement is undoubtedly sometimes an early symptom of the disease.

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* Although an occasional relation to syphilis had been noted by several preceding observers, Fournier was the first (in 1876) to assert the wide extent of this relation. His statements were received with doubt (because syphilitic patients constituted his field for observation), but they were confirmed from the neurological side by myself ('British Med. Journal,' March 1, 1879) and Erb ('Arch. f. klin. Med.,' July, 1879); while abundant corroboration has been since afforded. Many who at first doubted have been convinced by fresh observations. Facts collected without reference to the point at issue have proved useless, and freshly collected facts have thrown new light on the subject. The proportion of cases with previous syphilis necessarily varies according to the absolute frequency of syphilis, because the number due to other causes will be the same. Syphilis is rare among Hebrews, and hence smaller ratio of Jewish tabetics have had syphilis. The difference in the character of the lesion from that of changes known to be syphilitic was urged as an objection by many writers, but such considerations are theoretical, and must yield to facts; instead of denying that this or that lesion can be produced by a given cause, we may have to widen our view of the operation of that cause. Other degenerations of the nervous system seem also to have a relation to syphilis, as I pointed out some years ago ('Lancet,' Jan. 15, 1881). As will be shown in the section on Pathology, recent discoveries lessen very much the difficulty of comprehending the relationship.

Secondary Tabes.—Symptoms of locomotor ataxy sometimes succeed other diseases of the spinal cord, and such sequence is especially common in syphilitic subjects. Myelitis and syphilitic gummata may be thus succeeded by tabes. An officer in India, who had had syphilis, having suffered for a day or two from pains in the back, took a bath in snow water, and in a few days his legs were absolutely powerless. He gradually recovered power, but could not co-ordinate the movement: as power returned, lightning pains came on, and a year afterwards he presented the typical condition of locomotor ataxy. Again, a man, twelve years after syphilis, had a severe fall, followed by gradual loss of power, so that at the end of three weeks he could scarcely stand. The legs remained weak for a month, and then improved, but ataxy came on; three months later, power was good, inco-ordination extreme. A similar succession may occasionally be observed in those who have not had syphilis. In many cases, however, the ultimate condition is one of combined weakness and ataxy.

SYMPTOMS.—A typical case of developed tabes presents certain motor, sensory, and reflex symptoms. There is inco-ordination of movement of the legs, sometimes of the arms also, without loss of power or muscular wasting. There are pains in the affected parts, especially sharp momentary "lightning" pains; there is some loss of sensation; there is often loss or diminution of reflex action from the skin, and almost always entire loss of the myotatic irritability that is revealed by the so-called "tendon-reflexes," and especially by the knee-jerk; there may be retention or incontinence of urine, constipation, and often loss of sexual power. Of this group of symptoms, two usually precede the others—the pains, and the loss of the knee-jerk. These may exist alone, even for years, before inco-ordination comes on. Thus the symptoms are far wider in range than the name "ataxy" suggests; and while inco-ordination, if it exists, is the most obtrusive objective symptom, it may never be developed. Hence physicians have extensively fallen back on the older term "tabes dorsalis." But recent expansion of our knowledge, especially the discovery of the extent to which the symptoms may depend on disease of the peripheral nerves, shows that even the qualifying "dorsalis" narrows the name unduly, and "peripheral neuro-tabes" has been added to the terminology of the disease.

Besides the symptoms above enumerated, others are occasionally present. Of these the most important are atrophy of the optic nerve (and occasionally of other cranial nerves); trophic changes in the skin, the bones, and the joints; peculiar paroxysmal visceral disturbances and occasionally motor paralyses and muscular wasting supervene. The combinations of symptoms present in different cases vary much. It will be convenient to consider first the individual symptoms in their various degrees, and then their grouping and sequence.

Motor Symptoms.—The characteristic inco-ordination of movement develops gradually. It is usually increased (as Romberg pointed out) by closure of the eyes, and at first may only exist when the guiding influence of vision is thus withdrawn. Before it causes ataxy of movement, it may render difficult the maintenance of equilibrium when the base of support is narrowed by the feet being placed close together, toes and heels; if then the eyes are closed, the patient sways, and tends to fall. In health slight unsteadiness is thus produced, varying in degree in different persons, but never amounting to even a suggestion of a fall. The effect of closure of the eyes is greatest when sensation in the soles of the feet is defective, but does not depend on this defect; it may be marked when sensation on the soles of the feet is perfect. The early defect in co-ordination may be discovered by the patient when he walks in the dark, or backwards, or, not uncommonly, when he shuts his eyes in the process of washing the face. In a further degree of inco-ordination there is inability to stand with the feet together, even when the eyes are open, and the patient is only steady when the feet are wide apart. If the feet are bare, the difficulty is greater, because muscular action has to replace the rigid base of the boot. The irregular contraction of the muscles is shown by the conspicuous movement of the tendons on the back of the feet. The patient may oscillate from toes to heels before he comes to rest. As the defect progresses, uncertainty is felt in walking even with full visual guidance, especially on uneven ground, or on a very smooth surface. A slight visible alteration in gait is then appreciable, the feet are not placed on the ground quite as in health, or there is distinct difficulty in maintaining equilibrium when the patient turns quickly, and he has to put a foot down suddenly to keep from falling. As the inco-ordination increases, the change in gait becomes greater, but varies much in its precise characters, according to the muscles that are most affected. Often the feet are raised too high, thrown forwards too far, brought down too suddenly, and the whole sole comes in contact with the ground at once. There may be a tendency for one foot to be brought in front of the other, or moved too far outwards. Often the foot becomes inverted when it touches the ground. Efforts to correct error in movement have themselves to be corrected. In other cases the defect in maintaining equilibrium is greater than the actual disorder of movement of the legs, and the patient sways about in the manner of one who has cerebellar disease; this probably depends on a preponderant affection of the muscles of the hip-joint.

As the defect progresses, the patient is only able to walk by steadying himself with a stick, or by taking hold of the arm of another person or of adjacent objects. At first a very slight degree of this help is sufficient; guidance rather than support is needed. Afterwards, however, considerable support is necessary, and ultimately the patient may be unable to stand even with help. When he attempts to rise, the legs move hurriedly forwards and backwards, and if the upright posture is at last achieved, the legs slip forward, and only strong support saves the patient from a fall. The ataxy is manifest also in other movements of the legs. If the patient, when lying, tries to touch an object with his foot, the leg is moved irregularly, goes beyond the place, and then is brought too far back, and only at last does it come in contact with the object, often with unintended force. This inco-ordination, like that in standing, is much greater if the eyes are closed.

The arms may present similar inco-ordination, although they often escape, even when the affection of the legs is extreme; very rarely the arms suffer before the legs. The commencing defect is revealed by delicate movements, such as writing. When slight, it may be conspicuous if the patient tries, with closed eyes, to touch some object, such as his own nose, or, having abducted his arms, tries to bring the forefingers together. As it increases, all movements become irregular; it is impossible for the patient to button his coat, or to pick up a small object from the table; the fingers twist about in the attempt. The grasp is not sustained; first one finger is felt to relax and then another. If the patient attempts to hold out his hand in a fixed posture, it is seen that the same irregularity obtains; instead of a uniform balanced contraction the muscles contract and relax involuntarily, and slow unintended movements of the fingers result, sometimes closely resembling those of athetosis. The same spontaneous movements may also be observed in the legs. They cease at once when the muscular effort is relinquished. Occasionally the muscles of the trunk present inco-ordination. Thus one patient could sit steadily on a chair when his eyes were open, but if he closed them would at once fall off. The movements of the head, face, tongue, and eyes, escape the characteristic derangement.

Even with extreme inco-ordination, the power of the muscles may be unimpaired. Occasionally some group of muscles, as the flexors of the ankle, become weak for a little time and then strong again, just as may the eyeball muscles, as we shall presently see. In some cases motor power in the limbs remains unimpaired to the end; more often, when the ataxy has become great, some muscular weakness supervenes, with or without wasting of the weak muscles. There is a distinct group of cases in which weakness and ataxy come on together, but these are considered separately (" Ataxic Paraplegia ").

Sensory symptoms are prominent in most cases,—subjective sensations, especially of pain, and loss of sensibility. Spontaneous pains are present in some degree in nine tenths of the cases of tabes. The most frequent and characteristic are the sudden and lancinating pains called "lightning pains." They occur chiefly in the legs, but may be felt in the trunk, arms, and even in the head. They are usually paroxysmal and apt to come on at night; attacks of such pains last for some hours or for a day or two, varying in seat, but often felt in the same part throughout an attack, or through many attacks.

Sometimes they are felt in a limited area of the skin; at others they seem more deeply seated; sometimes they dart down the limb. They may correspond to a nerve-trunk (especially the sciatic), but more frequently have no relation to the nerves. They may be felt near but not in joints. Although the pains are usually acute and "stabbing," they are sometimes of different character. Thus in one patient the sensation was as if both legs were for a moment on fire. When referred to the skin, this often becomes tender after the pains have been felt for a time, and even the contact of the bedclothes cannot be borne. When pains continue at one spot for some days, vascular or trophic changes may occur there,—ecchymoses have been observed, and I have known the growth of the hair to be changed where pains had been felt in the scalp; each hair bent and broke short off, over an area the size of half-a-crown; after the pains ceased, the growth of the hair became normal. A sharp pain may be accompanied by a sudden reflex spasmodic movement of the legs, or by inhibitory weakness. Thus, in one case, a sudden pain would often make the patient fall on his knees. The pains may be so severe as to prevent sleep for several days.

Other kinds of severe pain, less brief than the lightning pains, occur in some cases, and are described by various epithets, as "burning," "tearing," "gnawing." Burning pain in the toes distressed one patient. Much more common, and occurring with other pains, are slighter dull pains like those of rheumatism, for which they are often mistaken, an error that is facilitated by the circumstance that any of the pains of tabes may be influenced by weather, being especially increased by damp cold. Such dull pains sometimes precede the sharp pains even for years. A painful sense of constriction is also common, and may be felt around the trunk or in the legs, groins, &c.; it is often referred to a considerable area—the patient may feel as if the whole trunk were tightly enveloped in a cuirass of brass. Darting pains also may be felt in the trunk and not in the legs. Such a sense of constriction is sometimes referred to the legs. In the arms and hands the pains are usually slight in degree, and seldom cause real distress. Visceral pains may also occur, usually paroxysmal, and referred to the stomach, bladder, rectum, &c.

Pains are not only the most common, they are often the earliest indication of the disease, and may be the only subjective symptom in slight and stationary cases. They bear no proportion to the other symptoms, and are sometimes trifling—occasionally altogether absent. They seem sometimes to occur alone; I have seen several patients who suffered lightning pains, characteristic in features, course, and general associations, both with and without the pupil symptom common in tabes. Such isolated pains may be followed ataxy.

Sensations other than pain are also common. They are various in character, described as "tingling," "pins and needles," "creeping," and very often as if the skin, especially of the soles, were covered by some soft substance. Sensations of cold or of heat are also common; the feet may feel as if the legs were always in cold water; a sensation of cold about the testicles is occasionally complained of. Similar cutaneous sensations are also common in the hands. Increased sensitiveness to pain may also exist, especially on the soles of the feet, and often accompanies lessened sensibility to touch.

A diminution of sensibility is also present in most cases of the developed disease. It may involve all forms of sensibility, but seldom equally; some forms may be impaired and others normal. Either pain or touch may be affected alone; for instance, a prick may cause no sensation of pain, although the least touch of the finger is everywhere perceived. A firm touch (*i. e.* slight pressure) may be felt when a slight touch is unperceived. When a touch is felt, the power of localising it may be impaired. Temperature sensations are rarely affected without other forms of sensibility, but they may be impaired with pain when tactile sensibility is normal. On the other hand, there may be extensive loss to pain without any defect of the temperature sense. A prick may then cause only a sensation of heat, and extreme degrees of heat or cold cause pain less readily than normal. The temperature loss may be partial—either cold or heat may be unperceived while the other is recognised. When touch is slightly impaired, there may be an inability to discriminate differences in the degree of pressure on the skin.

Accompanying the diminution or increase of sensitiveness there are sometimes curious changes in the sensation produced. One of these is a delay in the perception of pain, which may amount to several seconds. The greatest delay I have met with amounted to seven seconds, but one of fifteen seconds has been described (Eulenberg). The contact of a point may be felt at once, the pain only after a considerable interval. Often there is an after-pain, lasting sometimes for a quarter or half a minute or longer, and the maximum sensation may not be attained until some seconds after the pain is first felt. Thus, in a case in which there was a delay of seven seconds, the maximum sensation was only felt twenty-five seconds after the prick (Obersteiner). Often there is a second or third maximum, a sort of rhythmical recurrence of sensation, quick or deliberate. A delay greater than normal may also attend the sensation of heat. The localising power is sometimes strangely perverted. A touch or prick on one leg may be referred to the other, sometimes with singular uniformity in position (allocheiria, p. 9). A prick in one spot may be felt in many places (polyæsthesia) on the same, or on both legs. The reaction of the sensory nerves to electricity may be changed in the same way as that of the motor nerves when they are degenerated; instead of the earliest sensation, on closure of the circuit, occurring at the kathode, it occurs at the anode (Mendelssohn).

Impairment of sensation is most frequent, and usually greatest in the lower part of the legs and feet. It may even be limited to the soles, and usually then ceases at the outer sharp edge of the foot, but extends for an inch or so up the inner side. Touch or pain may be lost on the sole only, and even, strange to say, the form of sensibility that is lost on the sole may alone persist in the legs. Although the sole is usually most affected, sensation may be perfect on the sole when lost elsewhere. One leg is often more affected than the other. The loss may extend to the trunk, and over part or the whole of it. In the arms it is usually greatest on the hands; often commences on the palmar aspect of the fingers, and may be limited to this aspect of the hands, just as it is to the soles of the feet. Sensation may be lost on the trunk and not on the limbs—an important fact, because the anæsthesia may then readily be overlooked. On the head loss of sensation is rare and partial.

The loss of sensibility is not confined to the skin, but affects the deeper tissues also in many cases. The contraction of the muscles on electrical stimulation may be unfelt, and the muscles may be less sensitive than normal to pressure or forcible extension. It is only from intelligent patients that this can be distinctly ascertained. Sometimes, however, the muscles may be quite insensitive when cutaneous sensibility is little or not at all impaired. It is probable that the sensibility of the joints, fibrous tissues, and tendons is also involved. The position of the limbs in passive as well as in active movements may not be perceived. In slight cases this sense of posture may not be lost, but in severe cases it may be so absolutely abolished that the patient has always to look to see where his legs are. The anæsthesia may extend to the viscera when the trunk is involved. In consequence of the loss of sensation, grave injuries to the limbs, such as burns, may be unperceived, and visceral disease may be unattended with the customary pain. Pleurisy, for instance, may be absolutely painless.

But impairment of sensation is not invariable. There may be no loss even in patients who present considerable inco-ordination, and in those who suffer severe pains, as well as in those who have had none. But it is very rare to have no sensory defect when inco-ordination is considerable.

Reflex action from the skin is usually impaired in proportion to the loss of cutaneous sensibility, and especially to the loss of tactile sensibility. It may be normal when pain is unfelt. The plantar reflex is most frequently impaired, and the progress upwards of the disease may be attended by a progressive loss of the gluteal, cremasteric, and abdominal reflexes. On account of the variations met with in health, it is uncertain whether reflex action is ever lessened when there is no impairment of sensation. On the other hand, in the early stage of the disease, there is sometimes a considerable and even extreme excess of all cutaneous reflex action.

Loss of sexual power is an exceedingly common and often an early symptom of the disease, and may precede any impairment of sensory or reflex functions. On the other hand, exceptions are met with in which sexual power persists to a late period of the disease, even until after the patient has lost the ability to walk alone.

If the condition of cutaneous reflex action is somewhat inconstant, this is not the case with the reflex process on which the myotatic irritability depends. A loss of the knee-jerk, as Westphal first pointed out, is one of the earliest and most constant symptoms of tabes. With it disappear also all other indications of this irritability. In cases with great excess of cutaneous reflex action the knee-jerk is lost as in other cases, and the contrast between the two forms of reflex action is very striking. It is exceedingly rare for the knee-jerk to be obtained in any case of true tabes, but an early case is sometimes met with in which it is not lost, although diminished, and commonly unequal on the two sides. In one or two cases I have found it slight on one side and lost on the other, when distinct ataxy was present, and have observed its subsequent disappearance. In one case, in which the jerk was lost in one leg only, lightning pains were confined to this leg. The loss often precedes for many years the development of inco-ordination.

Eye Symptoms.—The functions of the internal muscles of the eye are often affected in tabes. In more than five sixths of the cases[*] the reflex action of the iris to light is lost; rarely the action remains perfect throughout. In most cases of reflex loss, the contraction on accommodation is preserved, as first noted by Argyll-Robertson. Often also (as Erb has shown) the dilatation that occurs on painful stimulation of the skin of the neck, &c., can no longer be produced. Sometimes the ciliary muscle is also paralysed, causing loss of accommodation. Very rarely there is loss of accommodation and of the associated contraction of the iris, without loss of the light-reflex. When reflex contraction is not lost, it may not be maintained; the pupil again enlarges; an actual inverted reaction to light and darkness has been observed.[†] The pupils are often very small ("spinal myosis"), especially when there is loss of the reflex dilatation from the skin, a loss which seems to be accompanied by atony of the radiating fibres supplied by the sympathetic. The pupils are not always small; they may be of a medium size or still larger, and then I have usually found that the skin-reflex can be obtained, although the light-reflex cannot. They are often not perfectly circular, and are frequently unequal. Both eyes are usually affected in the same manner; occasionally the reflex action is lost in one and only lessened in the other; indeed, almost every possible condition is occasionally met with. On the other hand, the intra-ocular muscles may be unaffected even in advanced cases. According to Berger, the ocular tension is often lessened.

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[*] Berger says 97 per cent.

[†] L'Abunde, 'La Psychiatria,' 1889, vii, p. 283.

Paralysis of the external ocular muscles is also common in tabes, and occurs in several forms: (1) Transient weakness, lasting a few days or weeks and then passing away. (2) Permanent paralysis, complete or incomplete, of a single nerve or part of a nerve. Either form may occur at any stage, but the first is most common in the early, and the second in the later stages of the disease. The transient palsy renders diplopia a common symptom in the early period; any muscle may be affected, but the external rectus is that which most frequently suffers. The persistent palsy may affect one or more muscles; often the levator is involved, causing what has been termed "tabetic ptosis" (Fig. 114). Sometimes the whole third nerve is paralysed. (3) There may be a combined palsy, suggesting a central origin, as, for instance, loss of the movement of convergence associated with loss of accommodation, although the internal recti act in the movement towards one side. (4) Many or all the muscles of both eyes may gradually become paralysed, "progressive ophthalmoplegia," internal or external—a condition that is more fully described in the account of ocular palsies in vol. ii.

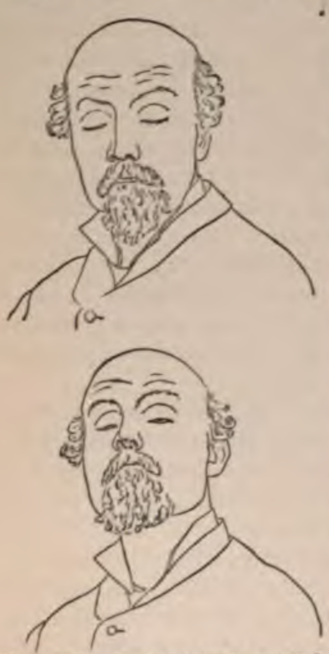

FIG. 114.—Double tabetic ptosis.[*]

Atrophy of the optic nerve is the most serious ocular complication of tabes. Its frequency is difficult to ascertain, but is certainly less than is often supposed; it does not occur in more than one case in ten. Atrophy is generally an early symptom, usually commencing before inco-ordination is developed, and in a large number of cases ataxy does not supervene; there seems a tendency for the spinal malady to become stationary when the optic nerve suffers. The disc becomes pale, and its substance shrinks, so that recession begins at its edge instead of at the central "cup," as may readily be perceived by the course of the vessels. The failure of sight usually commences with a peripheral limitation of the field and loss of colour vision, but sometimes central acuity fails early. When this remains good, the peripheral loss may reach a considerable degree before it is suspected by the patient. The visible pallor of the optic disc is not always proportioned to the failure of sight; the change in tint depends on wasting of the capillaries, and this does not always correspond to the wasting of the nerve-fibres. The ultimate tint is grey, or a whitish grey, especially as seen by the direct method. When the disc was very vascular before the atrophy, the grey tint is generally deepest. The vessels are but little narrowed. Occasionally there are slight signs of congestion in the early stage, and then some tissue of gelatinous aspect may develop in the disc, and the vessels may appear somewhat compressed.*

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[*] From a Salpêtrière photograph, for which I am indebted to M. Charcot.

The course of the atrophy is usually slowly progressive, and, in most cases, it ends in total or almost total blindness. It may, however, cease to progress, and even undergo slight improvement. The onset is not always gradual; sometimes sight fails considerably in the course of a few days, without ophthalmoscopic changes to account for it. I have met with one such case in which double temporal hemianopia indicated disease at the optic chiasma. In such cases the secondary interstitial process probably assumes inflammatory intensity (see p. 396). The atrophy is often more advanced in one eye than in the other, and very rarely one eye only suffers. Flashes of light are occasionally experienced in the course of the atrophy, comparable to the lightning pains in the limbs, but they are not common.

Deafness, having the characters of nerve-deafness, is met with in some cases, sudden or gradual in onset, transient or lasting. The persistent deafness has been attributed to an atrophy of the auditory nerve, analogous to that of the optic nerve, but certainly in many cases without sufficient reason.† Only when there is a progressive limitation of the range of hearing, analogous to the peripheral limitation of the field of vision, are we justified in assuming nerve atrophy. I have seen two cases of this kind. In each there was also optic nerve atrophy, and the other symptoms of tabes were distinct.‡ Attacks of vertigo are frequent in the cases attended by deafness, and seem to depend on the disturbance of the labyrinth or auditory nerve, and to be thus connected only indirectly with the primary disease. Anosmia occasionally occurs, and is probably not infrequent. It is apparently due to an atrophy of the olfactory nerve similar to that of the optic.

The functions of the other cranial nerves are not often affected. Pains may be felt in the region of the fifth nerve, and there is occasionally loss of sensation in some part of the area supplied by it, on one side or both. Unilateral atrophy of the tongue has been sometimes noted. In the larynx, besides the spasm to be presently described, tate amputation. The growth of the nails of the feet, and sometimes of those of the hands, may be changed; they may be thickened and the surface furrowed or irregular. The nails may even fall off and be slowly renewed. The teeth sometimes decay quickly, and may drop out. One patient who had previously lost many of his nails, found that all the teeth of the upper jaw fell out in the course of three days, without any pain or decay. Wasting of the muscles occurs only as a complication in the later stage of the disease.

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* Further details will be found in 'Medical Ophthalmoscopy.'

† It is very difficult to distinguish an affection of the nerve from one of the labyrinth independent of the nerve. Even in a case of bilateral deafness coming on in the course of tabes, in which the patient could hear only a loud voice, and deep notes better than high ones, in which atrophy had been diagnosed, Lucae found calcareous masses in each of the labyrinths, and the auditory nerves were quite normal.

‡ In one, only notes between E^3 and D could be heard. In the other the patient, when first examined, was absolutely deaf to notes above E^2 and below G_1. A few months later, the restriction had confined the range of hearing to the octave in the treble clef, between E^2 and E^1, even $E\flat^1$ being inaudible. Thus the loss occurred chiefly from below upwards. Ultimately no note could be heard.

Changes in the nutrition of the joints and bones have attracted much notice since attention was directed to them by the careful investigations of Charcot, whose name is sometimes connected with the condition. They are not very common, but are sufficiently frequent and well marked to place their relation to the disease beyond doubt. The bones may become brittle, and may break readily, in so-called "spontaneous" fractures. The process of union is attended with the formation of a large amount of callus. Sometimes there is also ossification or calcification of the structures adjacent to injured bones, and even of the fibrous tissues independent of the joints, such as the intermuscular and subcutaneous tissues. This may be rapidly inflamed, and extremely quick swelling may occur, followed by induration. As the inflammation subsides, there is a remarkable tendency for ossification to occur even in the newly-formed inflammatory tissue beneath the skin. Such subcutaneous changes occur most frequently in the foot or thigh, especially near joints, but sometimes in the deeper fibrous tissues between the muscles. In the joints, the changes consist of erosion of the cartilage, wasting of the heads of the bones, and ossification of the ligaments; sometimes irregular bony growths form, and occasionally the head or shaft of a long bone may undergo an extraordinary increase in size by the development of new osseous tissue on the surface. The joint changes may thus assume an atrophic or hypertrophic character, and the movement may be either too free or too limited. Wasting of the head of the femur often renders dislocation of the hip easy. A remarkable instance of these osseous changes has been recorded by Charcot; a tabetic woman suffered successively, in the course of a few years, dislocation of both hips, fractures of the left femur, of both bones of each forearm, and dislocation of one shoulder; after death old fractures were found in both scapulæ and the left clavicle. With such changes, and often when they are slight in degree, there may be great effusion within the joints and œdema outside them. The larger joints are those most commonly diseased—knee, hip, ankle, elbow,—but the small joints of the fingers have been known to suffer (Westphal). The relative frequency with which the chief joints are affected is approximately indicated by the following percentage:[*]—knee, 45; hip, 20; shoulder, 11; tarsus, 8; elbow, 5; ankle, 4. They often occur during the pre-ataxic stage. The lesions are sometimes excited by injury, and the extraordinary changes that may follow traumatic influences, or occur alone, afford a conclusive indication that abnormal trophic influences are at work. Fig. 116 represents the arm of a man who fell and struck his elbow, fracturing the olecranon and condyle. This was followed by extreme painless swelling, and bony masses could be felt, gradually increasing in size. After death the fractures of the bones were found, and masses of new bone had formed in the capsule of the joints. The arthropathic changes occur also, however, without injury, and are probably due to disease of the nerves, which not only has a direct trophic influence, but also renders the structures insensitive to the injurious effects of prolonged tension or pressure (Rotter). When inco-ordination has developed, the variations in the muscular contractions involve varying strain on the ligaments, especially in the case of joints, as the knee, that depend for part of their support on

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[*] Obtained by combining the collections of cases of Weizsäcker (Bruns' 'Beitr. z. kl. Chir.,' 1887) and Rotter ('Arch. f. kl. Chir.,' Bd. xxxvi).

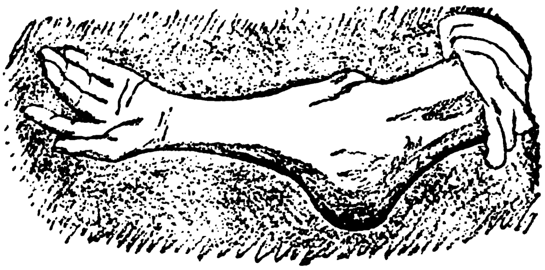

FIG. 116.—Locomotor ataxy, painless swelling of elbow-joint after a fall on it. One condyle of the humerus and olecranon were found, after death, to have been broken off. Several separate pieces of new bone had formed in the capsule of the distended joint.

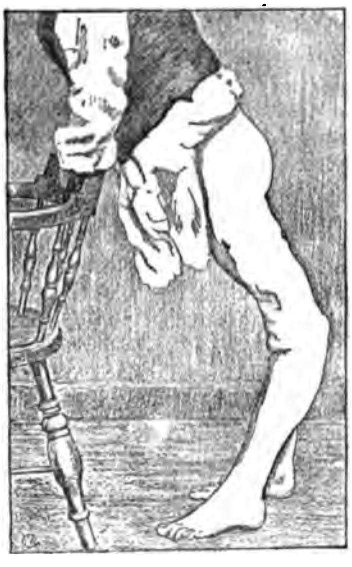

FIG. 117.—Locomotor ataxy; retroflexion of knee-joints.

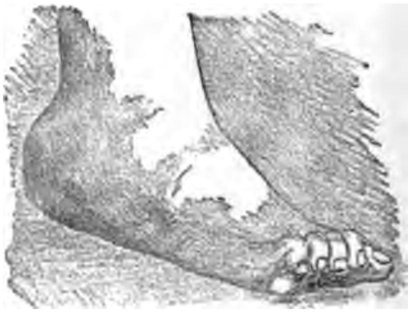

FIG. 118.—Tabetic foot (from a photograph by Mr. Marriott, B.Sc.).

the muscular tendons that pass by them. The knee-joint may thus become so lax that when the patient stands, the joint may become retroflexed, as in Fig. 117. Changes in the tarsal bones and articulations may cause the foot to become flat, with a projection inwards or backwards of the tarso-metatarsal articulations, and of the (often enlarged) tarsal bones. The condition has been called the "tabetic club-foot," by Charcot and Féré.*

Visceral symptoms of peculiar character occur in many cases of tabes.† They consist almost entirely of paroxysmal disturbance of function, usually attended with great pain, and have been termed, by the French, *crises*, with qualifying adjectives according to the seat of the symptom. There is a tendency to over-elaborate this terminology. The most frequent seat of such disturbance is the stomach, and these attacks are called *gastric crises*. They consist of paroxysms of severe gastric pain, felt in the epigastrium and often passing through to the back. The pain is accompanied by vomiting, with or without nausea. The vomiting is often incessant, and is first of food, then of clear liquid, which may be very abundant; ultimately bile is vomited, and sometimes blood, which may be altered in colour; it has been ascribed to simultaneous vaso-motor disturbance in the mucous membrane. Retardation or irregularity of the action of the heart has sometimes attended such an attack, and, rarely, pyrexia has been noted. I have known frequent hiccough to accompany the vomiting. Such an attack lasts for some hours or days, and then subsides, to recur in a few weeks. During the intervals the functions of the stomach may be performed in a perfectly normal manner. Although pain and vomiting usually occur together, some patients have attacks of pain without vomiting, and in others there is vomiting without pain. Rarely there is nausea alone. A temperate man, in the early stage of tabes, suffered during three years from frequent attacks in which, for several days, he had intense nausea each morning, passing away in the afternoon, and succeeded, in the evening, by an inordinate craving for food. He never vomited unless he made himself do so, in the vain hope of thus obtaining relief. After two years, attacks of laryngeal spasm were added to the nausea.

The chief *intestinal* disturbance is constipation, but paroxysmal diarrhœa has been supposed to be of vaso-motor origin, and connected with the disease (Pierret). Paroxysms of rectal pain (*rectal crises*) may occur, and may be accompanied by distressing tenesmus, sometimes by a sensation as if there were a foreign body in the rectum.

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* The trophic changes in bone and joint have been regarded by some as simple chronic osteo-arthritis, without causal relation to the nerve disease in their subjects, but the evidence is altogether opposed to this view. The arguments that can be adduced in support of it will be found in some of the speeches delivered at a discussion at the Clinical Society, Nov. and Dec., 1885.

† The occurrence of attacks of vomiting, sometimes early in the disease, was pointed out by Topinard, but thought by him to be a mere complication. (Topinard, 'De l'ataxie locomotrice,' Paris, 1864, p. 273.)

In rare cases, paroxysms of pain have precisely the character of attacks of renal colic (*nephralgic crises*), or are felt in the neck of the bladder or along the urethra or at the meatus (*vesical or urethral crises*). These may be attended by an intense desire to micturate, although the bladder may be empty and only a few drops of urine may be expelled. In one patient such pain was repeated several times an hour, and each attack lasted for several days. Blood may be passed after an attack.

Laryngeal crises are, perhaps, after those of the stomach, the most common. They vary much in character. The most common form is a true laryngeal spasm, with noisy inspiration and expiration, cough, and often considerable dyspnœa. The paroxysms may resemble those of whooping-cough or of laryngismus stridulus. Pressure on the superior laryngeal nerve at its entrance into the larynx, or on the trachea, or the introduction of a sound, will sometimes induce an attack. The spasm may last for a quarter of an hour or for some hours, but rarely continues so long as the gastric crises. In one recorded case the spasm spread to the pharynx, making swallowing impossible; a violent attack extended to the muscles of respiration, and the patient died asphyxiated. Death from these attacks is, however, extremely rare. Paroxysms of rough cough have been termed "*bronchial crises;*" in one case such attacks ceased when the patient began to suffer from gastric crises. These crises are often early symptoms, and they may continue for many years. I have seen a patient, still in the first stage, who gave a history of gastric crises during the preceding eighteen years.

Comparable to these paroxysmal visceral disturbances, although very different in character and more alarming in aspect, are cerebral symptoms; but these are, fortunately, very rare. They resemble the attacks that are common in general paralysis of the insane, and consist of transient apoplectiform seizures, of transient hemiplegia, or of convulsions, general or one-sided. They may occur early in the course of the disease. The alarm they occasion is not without foundation, since death has been known to occur during an attack of apoplectic aspect. Occasionally an actual lesion occurs (probably thrombotic softening), causing persistent hemiplegia. Paroxysms of vertigo sometimes seem to be of similar nature, but they more often depend on derangement of the auditory nerve or labyrinth.

Course and Termination.—It is convenient to divide the course of the disease into three stages:—(1) In which there is no alteration in gait, the common indications of the malady being the loss of the knee-jerk and the pains, often associated with loss of the light-reflex of the iris, and unsteadiness on standing with the feet together and eyes shut. (2) That in which there is distinct affection of gait, slight or considerable, but in which the patient is still able to walk, alone or with the aid of one or two sticks. (3) In which walking is possible only with the aid of another person, or it is impossible for the patient to walk or to stand. The first stage is not always present; inco-ordination may be developed simultaneously with the loss of the knee-jerk, as one of the earliest symptoms.

The course of the symptoms is extremely variable. The inco-ordination usually supervenes gradually in the course of months, but it sometimes develops very rapidly. I have known it to become extreme in the course of a fortnight from its onset, after the first stage had existed for a considerable time. The epithet "progressive," given to the malady by Duchenne, expresses a characteristic of a large proportion of the cases in which ataxy is developed, and he knew no other cases of the disease. The inco-ordination, once developed, often increases, slowly or rapidly, until it reaches a moderate or a considerable degree, and the sensory loss shows a like tendency. The pains seldom correspond to the other symptoms in degree or course. They are often very severe in the early period of the disease, and may either lessen or continue unchanged as the loss of sensation develops. Their persistence, even in severe degree, does not show progress in the disease; once set up, they may persist for years without any increase in the other symptoms, and seem in this to be analogous to neuralgia, being evidence only of functional activity in the altered nerves.

The power of recognising the first stage of the disease, which we owe to Westphal's discovery of the loss of the knee-jerk, has enlarged, and in enlarging has to some extent modified, our conception of the general tendency of the disease. It is exceedingly common for the first stage to remain stationary for a long time, for ten, twenty, and even, in one case, for twenty-five years, if the duration of the lightning pains may be accepted as proof of its existence. When all cases are taken together, and if the patients are subjected to careful treatment, I think that the disease shows a progressive tendency in less than one half of the cases.

The other symptoms also do not always progress *pari passu*. Sensibility may fail although inco-ordination does not increase. Conversely, the ataxy may become greater although sensation remains the same, and even when it shows a distinct improvement. To this we shall return in considering the pathology of the disease. Thus a patient had slight ataxy, loss to pain on thighs, legs, and trunk, and no loss to touch or temperature. At the end of a year and a half pain was perceived on the thighs, and sensibility to touch, and temperature were still normal, although the ataxy had increased. So marked, indeed, is the contrast between the course of different symptoms in some cases that it suggests a certain alternative tendency. We have seen that if optic nerve atrophy develops, the spinal symptoms often remain stationary, and this is also true of visceral crises; I have known gastric crises to cease when inco-ordination came on. In one patient all the symptoms of tabes passed away, optic nerve atrophy ceased to advance, and even the knee-jerk returned, when the patient became insane.

The rate of increase, when this occurs, varies much. Sometimes it is so slow that the ataxy is considerable only at the end of several years. On the other hand, it may be so rapid that in a few months the patient is scarcely able to walk. More commonly the course is variable; periods in which the disease is almost stationary alternate with others in which the symptoms increase rapidly. The exacerbations may seem spontaneous, or may be distinctly excited by some prejudicial influence—a chill, a fall, or some excess, alcoholic or sexual. They are sometimes very acute; a marked change may occur in the course of a few days, or even in a few hours. Thus I have known a patient to pass, in the course of twenty-four hours, from a condition in which he could walk fairly well to one in which he could scarcely stand. Muscular power may lessen in such a sudden exacerbation, or the increase may be confined to the special symptom of the disease—the ataxy. But all such cases need re-investigation in the light of the facts that have been ascertained regarding the participation of the peripheral nerves in the process. An acute exacerbation may be "parenchymatous" in nature, seated in the elements either of the spinal cord or peripheral nerves. The great difficulty of the problem is due to the fact that there may be a myelitic or neuritic complication, or merely an acute process in the nerve-elements in which the chronic disease was in progress, and similar in nature. It may be extremely difficult to determine which of these is the cause of the rapid increase in the symptoms. The conditions thus developed may pass away again, leaving the patient on a little lower level than before, or they may persist without any considerable recovery.

There is nothing in the nature of the disease, in most cases, to produce death. The only direct effect of the malady, which has ended life, is laryngeal spasm or paralysis. Even the gastric symptoms, prostrating as they are, are never fatal. Patients who have reached the third stage have lived, bedridden, for twenty years. Death often results from intercurrent maladies, some of which are more dangerous to these sufferers than to others because they may develop painlessly, and attain a serious degree before their existence is suspected. But many patients die from indirect results of the disease. Kidney complications are the most common; they often develop insidiously, and only manifest themselves when life is in peril, as, for instance, by acute febrile symptoms, the exact nature of which may be unsuspected until the urine is examined. I have known a patient, still in the first stage, die thus after a few days' illness. Bedsores and pyæmia occasionally cause death, although far less frequently than in most other diseases of the spinal cord.

Complications.—Occurring, as most cases do, in syphilitic subjects, tabes is sometimes complicated by true syphilitic lesions of the brain or cord, generally the later lesions, such as gummata, or sudden palsy from the disease of a cerebral vessel.

Acute or subacute myelitis may occur during the course of the disease with characteristic symptoms. Such a complication must not be confounded with the simple acute exacerbation in the special symptoms, which sometimes occurs with remarkable rapidity.

Other system diseases of the cord may develop as complications of tabes. The lateral columns may be diseased with the dorsal posterior columns, giving rise to "ataxic paraplegia," but this is a distinct affection, and will be so described. Weakness may, however, be present from the first, or may supervene slowly, in the course of tabes, without muscular atrophy, and with a persistence of the special symptoms of the disease, from extension of the morbid process to the lateral columns. Another occasional complication is muscular atrophy, both the local atrophy already mentioned (and especially frequent in the tongue) and general atrophy, such as is met with in the pure spinal form. Thus a gentleman, who had had hemiplegia from syphilitic vascular disease, developed the first stage of tabes—pains, loss of knee-jerk, loss of the iris-reflex, and slight unsteadiness. He had two courses of mercurial treatment at Aix-la-Chapelle, and at the end of the second, atrophy began in the muscles of the hands, and spread during two years until almost all the muscles of the arms, shoulders, and back became extremely wasted. Local muscular wasting (according to the researches of Déjérine*) is usually the result of degeneration of the peripheral nerves; it is generally symmetrical. The extensive wasting, as in the case just mentioned, is probably due, as a rule, to degeneration in the anterior cornua.

In rare cases the legs present the symptoms of tabes, while in the arms there is the jerky inco-ordination of disseminated sclerosis, and the co-existence of the two lesions has been demonstrated.

Another very important and frequent complication of tabes is general paralysis of the insane. The two diseases have many alliances. It is probable that syphilis predisposes to general paralysis, as well as to tabes. Reflex iridoplegia is common in both diseases. The two maladies are often combined, and the symptoms of one or the other may preponderate. Thus many general paralytics present symptoms of tabes, and its characteristic lesion is found after death. On the other hand, cases of tabes may present slight symptoms of general paralysis, perhaps only slight optimism and mental weakness, which may remain subordinate, or may increase to a pronounced and preponderant degree. It may be difficult to say in which category a case should be placed. It is sometimes said that the disease may commence as ataxy, and may change to general paralysis, but a more correct expression of the facts is the co-existence of the two affections, and the dominance of the symptoms of one or the other.

Of complications of tabes outside the nervous system, besides the indirect consequences of the malady, valvular heart disease is the most important, and usually develops without any of the usual antecedents, and at an age at which degeneration is unlikely. It is not improbable that the coincidence is really one of cause, the valvular disease being the result of a syphilitic process. Other complications are so rare as only to deserve notice when a causal connection can be traced or reasonably suspected. Coincident glycosuria has been ascribed to an affection of the medulla oblongata (or may be produced through the cervical spinal cord), but it is extremely rare.

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* Déjérine, 'Revue de Méd.,' 1889.

PATHOLOGICAL ANATOMY.—In most cases the spinal cord presents changes visible to the naked eye. The posterior columns have a grey translucent appearance, which is due to the loss of the nerve-fibres and to an increase of the connective tissue ("grey degeneration"). The whole of the posterior columns may be thus changed, or only parts of them, the distribution of the visible alteration being that of the histological changes revealed by the microscope and presently to be described. In the hardened cord the difference of tint in the diseased areas is even more distinct than it is in the fresh organ. The posterior columns are also smaller than normal, because the bulk of the connective tissue is less than that of the normal fibres: hence the shape of the cord is slightly changed.

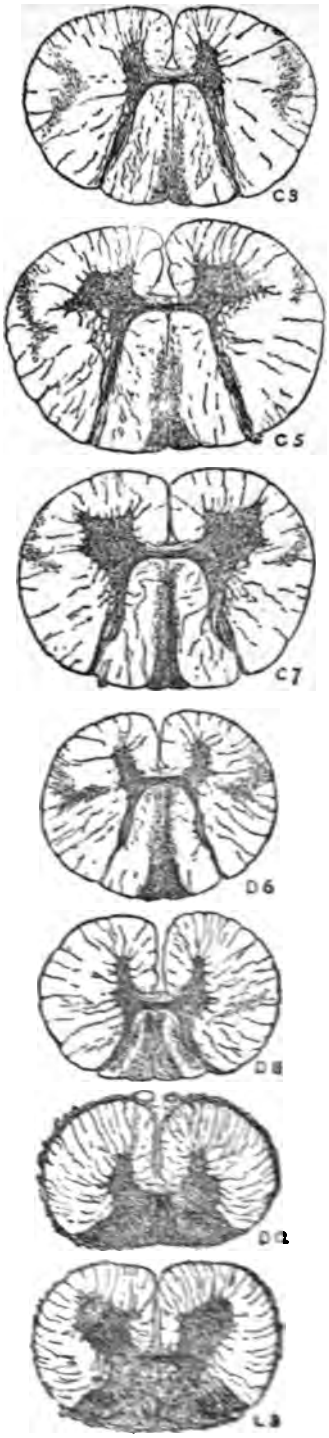

FIG. 119.—Locomotor ataxy involving the legs only. Sclerosis of the whole posterior columns in the lumbar region, gradually becoming limited, in the lower dorsal region, to the root-zone and posterior median column. The latter only is affected in the upper half of the cord (ascending degeneration). There is also conspicuous degeneration in the antero-lateral ascending tract. This is rather greater on the left side, while the post.-median degeneration is greater on the right, in harmony with the fact that the latter consists of fibres which do not decussate.

In a section of the cord stained with carmine or similar reagent, and examined under the microscope, the affected areas are conspicuous by the deep staining of the connective tissue. The position of the excess of this tissue, of the "sclerosis," as it is termed, indicates the place in which the nerve-fibres have degenerated. In the most frequent condition, in which the legs only are affected, the sclerosis occupies the whole of the columns in the lumbar region (Fig. 119), but it is often slight in the anterior parts of the postero-external columns, which may even be free from sclerosis; also in the middle of these columns many more nerve-fibres may be seen than elsewhere. The sclerosis is most dense in the part adjacent to the posterior cornu, through which the posterior root-fibres run, and near the surface of the cord (Fig. 119, D 12). Above the lumbar enlargement the affection of the postero-external columns gradually ceases, but the degeneration is intense in the postero-median columns, and has the distribution of an ascending degeneration, as it in fact is, receding from the commissure in the upper cervical region. (Compare Fig. 119 with Fig. 74, p. 177.) In other cases the external band of sclerosis, adjacent to the cornu, continues separate through the dorsal cord and even through the cervical enlargement (Fig. 120); the median degeneration then extends up to the commissure throughout the cervical region, just as does the secondary degeneration that results from a cervical lesion (see Fig. 76, p. 178). But in the cervical enlargement, the unaffected area in the anterior part of the postero-external column is usually much larger than in the lumbar enlargement.

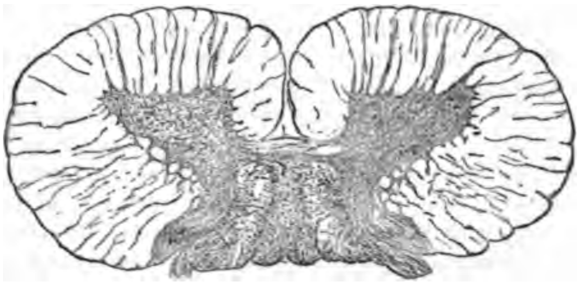

FIG. 120.—Tabes with ataxy of the arms as well as the legs. Section of cord in cervical region, showing sclerosis of the post.-med. column and root-zone of post.-ext. col. Degeneration of left ant. cornu.

In rare cases, of severe and long duration, the posterior columns are occupied by connective tissue in their whole extent from one end of the cord to the other. A few fibres of the posterior roots and a few vertical fibres near the commissure may alone be recognisable. An instance of such almost complete sclerosis is shown in Fig. 121.

On the other hand, in slight cases, in which the disease is still in the first, or in the commencement of the second stage, the sclerosis is moderate in degree, even in the regions of the cord most affected.

The separation of the sclerosed areas in the postero-external and the postero-median columns may be distinct throughout the cord, except in the lumbar enlargement, where the median degeneration expands into the external column. The sclerosis of the postero-external column is then usually limited to the root-zone, and varies in width, according to that of the area through which the root-fibres pass. In the dorsal region there is usually a narrow band of sclerosis close to the posterior cornu; in the lumbar region it reaches almost to the median septum. In some instances, however, a very slight degree of sclerosis extends from the most affected tracts through the rest of the columns, and is generally greatest in their posterior half. This condition existed in the very instructive case shown in Fig. 122. The intensification of this diffuse sclerosis in the root-zone and median columns is very distinct, although the affection of the latter is unusually slight. There is, moreover, in the dorsal region, sclerosis of another part of the postero-external column—the comma-shaped tract of short fibres, which degenerates downwards for a few inches below a transverse lesion of the cord (see p. 181 and Figs. 76 and 106, F). It is most distinct in 122 at D 8, but can be traced up to the cervical region, where its form is changed.

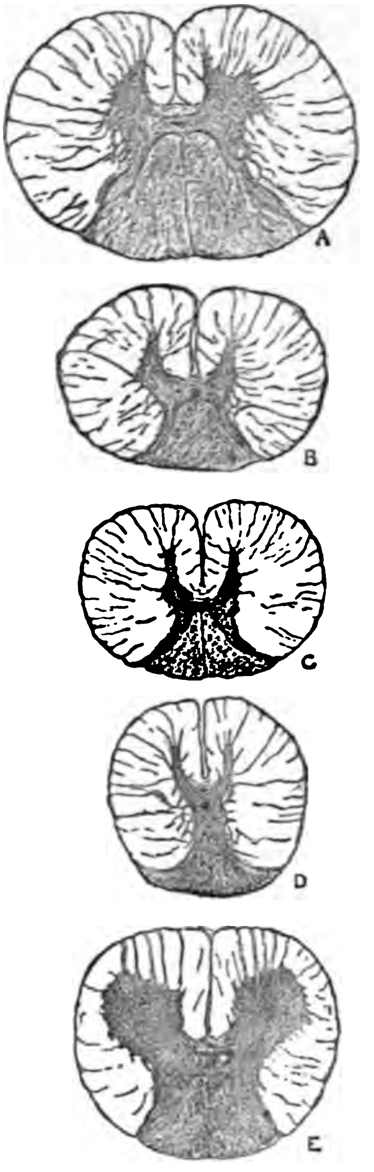

FIG. 121.—Locomotor ataxy: extreme inco-ordination and anæsthesia in both arms and legs; the posterior columns are sclerosed throughout the cord in their entire extent. A, upper cervical, B, C, D, dorsal, E, lumbar regions. In D there is also some degeneration of the intermediate grey substance.

The tract of fine fibres of the posterior root, discovered by Lissauer, is generally diseased.[*]

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[*] It was first described by Lissauer in connection with its disease in tabes;

The antero-lateral columns may be perfectly normal, even when the disease of the posterior columns is great. Often, however, in old cases, there is a slight general increase of the connective tissue throughout the cord. Sometimes there is distinct sclerosis of other definite tracts, *e. g.* of the ascending antero-lateral tract (as in Fig. 119), or of the direct cerebellar tract. In rare cases there is a distinct degeneration of the pyramidal tracts. The whole periphery of the antero-lateral column is thus occasionally the seat of sclerosis, the connective tissue extending in from the pia mater, which is usually also thickened; it is probable that this condition may be solely the result of the

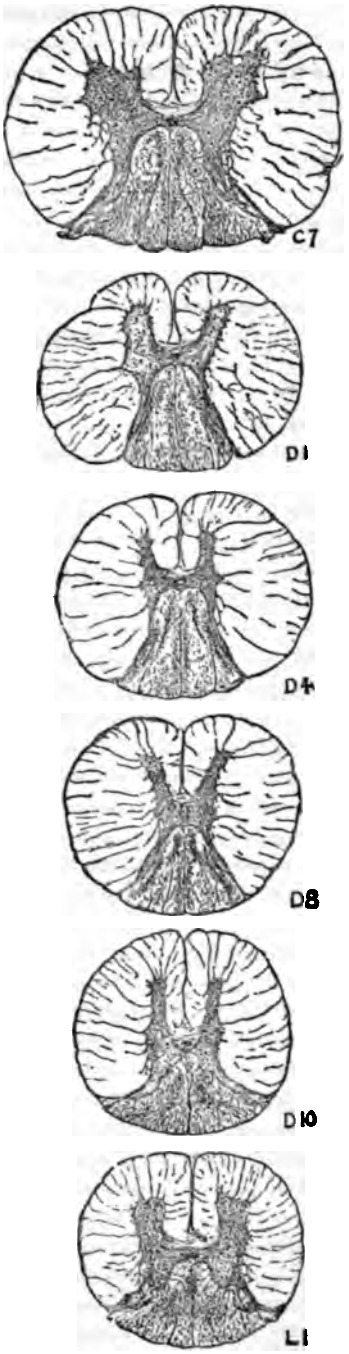

FIG. 122.—LOCO. ATAXY; SCLEROSIS OF POSTERIOR COLUMNS.—In L 1 the posterior portion of both post.-med. col. and post.-ext. col. is densely sclerosed, but in front only the post.-med. col. At D 10 the sclerosis is also general, but is dense only in the root-zone of the post.-ext. col. At D 8 and D 4 the change is slight, except in three areas, the root-zone, the middle part of the post.-med. col., and the comma-shaped tract in the front of the post.-ext. col. At D 1 the comma-shaped tract is narrow, and in C 7 it merges in a band of sclerosis, which bounds the anterior part of the post.-med. col. There is a little diffuse sclerosis through the rest of the column, and in the first dorsal there is some degeneration of the anterior cornua.—Symptoms of the first stage had existed for four years (lightning pains, slight analgesia, loss of knee-jerk and iris-reflex, gastric crises, optic nerve atrophy). A few weeks before death, the legs gradually became paralysed, and the arms ataxic; retention of urine caused acute kidney mischief, which was the immediate cause of death. No lesion was found after death to explain the subacute paralysis, but the peripheral nerves were not examined, their disease being then unknown.

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see 'Neur. Cent.,' 1885 p. 245; also ib., 1886, p. 419.

primary degeneration of the nerve-fibres even when it closely simulates the aspect of meningitic invasion.

In slight cases it is difficult to distinguish any changes in the grey matter of the cord, although it is probable that there is some atrophy of the nerve-cells and fibres in the posterior horn in many cases. Various structural changes can be discerned in more advanced cases (as Lockhart Clarke first showed), and the posterior commissure may be reduced in size. The posterior vesicular column also shows distinct degeneration in many cases. Some atrophy of the plexus of fine fibres contained in this column is said to be almost invariable (Lissauer), but frequently the cells and vertical fibres waste, and sometimes only a few shrunken cells, scattered through a translucent nucleated tissue, may remain. The atrophy of the cells is said to be especially associated with sclerosis of the direct cerebellar tract; also (but on slighter evidence) with that of large fibres in the pyramidal tract, which do not degenerate downwards. When the cells are unaffected, although the intervening substance is diseased, the cerebellar tract may be normal, and the fibres passing to it from the front of the vesicular tract may be unaffected. The degeneration of the grey matter often extends into the intermediate region between the cornua (Fig. 121, B); the intermediate lateral tract of cells may be atrophied, and occasionally even the anterior cornu and its cells suffer in a similar way (Figs. 120, 122, D 1). As we have seen, this change may be the cause of muscular atrophy, although in other instances the muscular wasting depends on alterations in the motor nerves.

The sclerosis varies considerably in its histological characters. In slight cases, there is merely a moderate thickening of the trabeculæ and their branches. The larger tracts are more fibrous than normal, and more nuclei are seen than in health. Everywhere the new growth of connective tissue proceeds chiefly from the pre-existing tracts, vessel-walls, &c. When the change is considerable, the trabeculæ are very broad, and areas seem to consist wholly of connective tissue, fibrillated and nucleated. In old cases there is sometimes little appearance of fibrillation in the most affected region; cells and thickened vessels lie in a granular or homogeneous tissue. From the parts most changed, the nerve-fibres have wholly disappeared; in other parts, however, close examination reveals many fibres narrower than normal, and many stained points that may possibly be axis-cylinders. The walls of the vessels are usually increased in thickness, sometimes to a very considerable degree. The coat next the endothelium may be thickened and contain many nuclei, but the chief increase, in most cases, is in the outer coat and adventitia, whence the tracts of tissue pass off into the adjacent region. The pia mater is sometimes thickened over the posterior columns; the vessels in it may then be similarly changed. The alteration in the membrane may extend over the neighbouring part of the lateral column, and even around the cord (Fig. 123), and, as already stated, septa passing from it into the cord may be thickened in the circumferential zone. This peripheral sclerosis is probably the result of three distinct processes, the relative share taken by each varying in different cases: (*a*) a primary degeneration of the peripheral ascending tracts; (*b*) a process of chronic interstitial myelitis, spreading from the pia mater, which is the seat of an inflammatory tissue formation; this may cause (*c*) a secondary ascending degeneration in the fibres affected in the first process. It is important to recognise this complexity of mechanism.

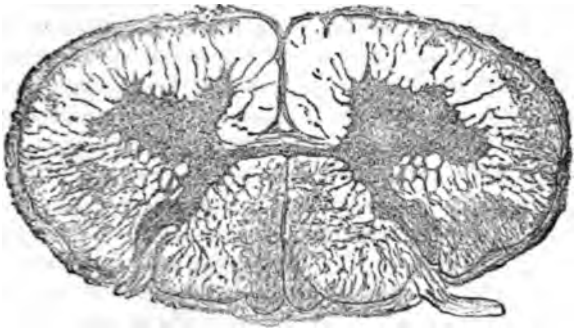

FIG. 123.—Tabes. Chronic meningitis. Cervical region. Sclerosis of post.-median columns and slight diffuse sclerosis of the cord, least in ant. cols., dense in the right lateral column. Thickened pia mater, and damage to subjacent superficial layer of the cord.

In cases of rapid course, products of degeneration, masses of myelin and granule-corpuscles, may be found in the affected parts, when these are examined by appropriate methods.

In rare cases other lesions are found in the cord, corresponding to the complications already described. The lateral columns may be sclerosed apart from any thickening of the membranes. Diffuse myelitis may be met with, and, occasionally, characteristic insular sclerosis has been found in the cervical region.

The posterior nerve-roots may appear normal in slight cases; it must be remembered that only a considerable degree of alteration can be detected even by the microscope. Often their disease is conspicuous; when the cord is much affected they are invariably affected, and may be atrophied, grey, and thin, to the naked eye, while the microscope shows wasting of the nerve-fibres and slight increase of connective tissue. The changes extend to the ganglia, where they cease. These structures are almost always normal, and the mixed nerve immediately beyond them is usually free from degeneration. The anterior nerve-roots are altogether normal, except in the rare cases in which the anterior cornua have suffered, and then some of their fibres may be degenerated, and the atrophied fibres may be traced also in the mixed nerve-trunk beyond the junction of the roots.

The peripheral spinal nerves have been found degenerated and in various degrees of atrophy in a large number of cases of tabes.* The change consists in a wasting of the nerve-fibres, beginning in the white substance, which may be reduced to a very narrow layer. Ultimately the axis-cylinders perish; they are said to present irregular swellings when the sheath begins to waste.† There may be a slight increase in the interstitial tissue and nuclei, but the change commences in the nerve-fibres themselves. The degeneration is greatest in the terminal nerve-filaments, and lessens in degree as the nerves are examined higher up, gradually ceasing in the larger trunks, and before the plexuses or corresponding main divisions are reached, the fibres are almost always healthy. The sensory fibres seem to be exclusively affected. The lesion has hitherto been found chiefly in the sensory nerves that supply the skin and joints, but that the sensory nerves of muscle undergo a similar change has been ascertained by Déjérine. It is probable that these afferent muscle nerves are invariably diseased, especially the fibres concerned in conducting the impressions that guide co-ordination, spinal and cerebellar,—on the former of which the knee-jerk depends, according to the theory maintained by the writer. The detection of this degeneration, among the unchanged motor fibres of the nerves, is very difficult, and this explains the paucity of evidence of it. The peripheral degeneration is found most commonly in the legs, but is met with also in the arms when these are the seat of symptoms. The change in the nerves bears no relation to that in the spinal cord in degree, or even in existence, for extreme alterations have been found in the nerves when the cord was quite normal. On the other hand, in some cases, the nerves have been healthy.

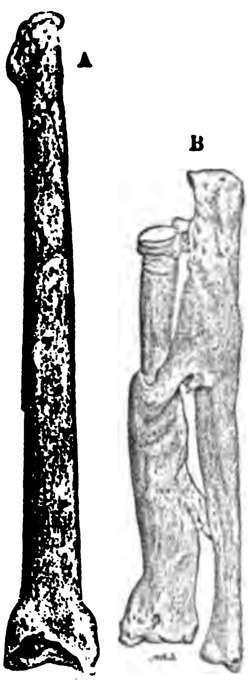

FIG. 124.—Osseous lesions in tabes. (After Charcot.) A. Atrophy of the head of the femur. B. Excessive formation of callus after so-called "spontaneous" fracture of the ulna and radius.

The optic nerves, when atrophied, present wasting of the nerve-fibres, and usually a remarkable increase in the interstitial connective tissue, which constitutes thick tracts, of gelatinous aspect. Degeneration of the ascending or descending roots of the fifth nerve has been repeatedly found (first by Westphal in 1864) in cases in which symptoms were present in the area of its distribution. Similar changes in the roots or nuclei of other cranial nerves have also been found.

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* The first observation was made by Westphal, but the extent and significance of the change in the nerves was pointed out by Pierret (1880), and his observation was soon after confirmed and extended by Déjérine and Pitres.

† Schmaus, 'Deut. Arch. f. klin. Med.,' 1890.

Pierret has described, as common in cases with vaso-motor and visceral symptoms, a degeneration of the intermedio-lateral tract and adjacent nerve-fibres in the upper part of the cord, traceable up the medulla oblongata in the region of the so-called "slender column," adjacent to the accessory, glosso-pharyngeal, and pneumogastric nuclei, from which it is supposed that the chief visceral and vaso-motor influences are exerted. The sympathetic has been examined in several cases, and found healthy, with one exception, in which the sympathetic on the left side of the neck was degenerated in association with left exophthalmos.

In the diseased bones or joints conspicuous changes are found after death. There is occasionally an extraordinary wasting of the articular ends of the bones, which seems to begin by rapid erosion of the cartilages, extending quickly to the bone beyond, the extremity of which may be much reduced in size and altered in form. In the hip-joint, for instance, in a case recorded by Charcot, the edges of the acetabulum had been removed, and the whole of the head of the femur and most of the trochanter had disappeared (Fig. 124).

Similar changes have been found in most of the larger joints. In the bones, the compact tissue has been found thinner and more porous than normal. At the seat of old fractures a large amount of bony callus is formed (Fig. 124). and sometimes new bone is found in the capsule of joints that have been injured.

PATHOLOGY.—The great fact of the pathology of tabes is that it is a neural degeneration in the sensory nervous system, peripheral and central. In both peripheral nerves and spinal cord, the incidence of the disease is almost exclusively on the structures concerned in afferent conduction. The cerebral system suffers in far less degree than the spinal, and in more irregular manner, but in the occasional peripheral degeneration of the optic nerve, and the central changes in the roots of the fifth, we may trace the same law of distribution. It is less dominant, however, than in the spinal system, since some of the cranial nerve symptoms are exclusively motor. The motor character of the leading symptom, ataxy, is, as we shall see, only an apparent and not a real exception to the general sensory character of the malady.

The degeneration commences in the nerve-elements themselves; the overgrowth of connective tissue, which gives to the lesion its obtrusive character, is secondary. This fact has been observed in the peripheral nerves; as regards the spinal cord it rests on inference scarcely less certain. That it is true of the spinal cord has, indeed, been doubted, chiefly on the ground of the thickening of the walls of the vessels seen in some cases, and the manner in which the increase of tissue seems to start from them. But the "system" character of a disease, the limitation of a wide-spread lesion in its early stage or slight degree to structures having a common function, is probably in all cases proof of its neural origin, i. e. its origin in the nerve-elements themselves. In tabes the evidence thus afforded is corroborated by the disease of the peripheral nerves, which unquestionably commences in the nerve-fibres, and by the partial character of the sensory loss, which means the limitation of the chief lesion to fibres of one sensory function. There is every gradation, moreover, between the cases in which the perivascular disease is great, and those in which it is so slight that there is nothing to suggest a primary interstitial process. The change in the vessels may reach a high degree in the posterior median columns when their degeneration is purely secondary, and certainly commences in the nerve-fibres; a similar thickening of the vessels has also been met with in secondary degenerations in other parts. It is most important to remember that the overgrowth of the neuroglia is a pathological process of growth distinguishable (and to a large extent distinct) from the degeneration of the nerve-fibres that excites it. It may vary in degree in different cases, and, even in the secondary degeneration of peripheral nerves, may assume an independent inflammatory character. Once excited, the energy of tissue-growth may be in some degree independent of its cause. Secondary vascular disturbance may attend it, and thus a sub-inflammatory condition (or even true inflammatory condition) is probably sometimes developed, which may invade the pia mater, and through this may spread widely.

The chief causal fact regarding the disease is its common relation to syphilis. Tabes is generally, although not invariably, a post-syphilitic disease; certain elements of the nervous system degenerate in consequence of the influence on them of previous or present syphilis. But these elements have a special liability to degeneration, which causes their nutrition to fail from other causes, and may also constitute their special susceptibility to suffer from the virus of syphilis. The usual symmetry of tabetic lesions indicates that the mechanism by which syphilis affects the nervous system is a blood-state. The manner in which the peripheral nerves suffer in many cases, brings tabes, in spite of its chronicity, into analogy with the more acute forms of peripheral neuritis, such as succeed acute specific diseases at a variable interval. The long interval, moreover, and the common slowness of the process in tabes when a sequel of syphilis, are only in proportion to the chronicity of the causal malady, compared with the acute specific diseases that have neuritic sequelæ. Further, the considerations mentioned in the account of multiple neuritis, which suggest that its immediate cause, when it is due to an acute blood disease, is a product of the growth of the organisms of the primary malady, rather than the organisms themselves, hold good also in the case of syphilis and tabes. The degenerative changes in the nervous system differ from the lesions of active syphilis, both in character and in time, in such a way as to make the assumption reasonable that they depend upon some product of the growth of the syphilitic organisms, a product which may possibly be a chemical substance—a suggestion first made by Strümpell, and widely held regarding other analogous maladies. Recent research is bringing into ever greater prominence the important part played by such products in the generation and course of diseases. In the specific diseases that have these sequelæ, there may be more than one such product of their organisms. causing more than one kind of consequence. The differences between such nerve degenerations as those of tabes, and other late effects of syphilis, is at least not difficult to comprehend on this hypothesis; neither is the fact—which has been so great a difficulty to many—that the treatment which has so speedy an effect on syphilis itself, is generally without influence on the degenerative processes that sometimes follow it.

Tabes is probably a rare sequel, when regarded from the side of syphilis. But the occurrence of such consequences in some instances, and not in others, has analogies in the case of almost all the acute specific diseases that have similar sequelæ, and must be taken as evidence of variations in the precise character of the primary disease. The virus of the primary malady is doubtless organismal, and such variations are familar to us in the case of other diseases. They sometimes have, and at other times have not, consecutive effects upon the nervous system, as in the familiar instance of diphtheria. The variations in the character of the organisms, and in their effects, may be relatively great, although entirely beyond our present powers of observation.

These considerations receive a notable emphasis from the occurrence of tabes in children who are the subjects of inherited syphilis, to whom the disease in early life is probably confined. The fact also shows how potent must be the part played by the agent, compared with the susceptibility that is, as it were, the causal complement—a fact seen also in the raritv of cases of tabes in which syphilis can be absolutely excluded.

The pathological facts already known enable us to understand many of the symptoms of the disease. There may be an interruption of the sensory path in one or both of two places, in the peripheral nerves, and in the posterior root-fibres as they enter the cord, and an interruption of the fibres in either place will explain the loss of sensibility which is so frequently present. The pains may reasonably be ascribed to the molecular changes in the nerve-fibres and their altered function, either in the periphery or in the cord. The intensity of a sensation is no measure of that of the process that causes it. Whether the affection of the sensory cells of the cord* takes part in the production of the anæsthesia and pains, we cannot tell. It is noteworthy that the affection of the peripheral nerves must involve the structures by which the nerve-impulses of sensation are normally generated by mechanical and other processes. The long persistence of pains without any increase in the symptoms, shows that they may be due to the action of structures that are changed, but not changing. The loss of reflex action from the skin is explained by the interruption of the sensory path, and an increased irritability of the sensory nerves, the result of the commencing degenerative changes, sufficiently explains both the hyperæsthesia and the increase of reflex action sometimes observed. Whatever theory of the nature of the so-called "tendon-reflex" action is held, the loss of the knee-jerk must be explained by an interruption of the sensory path. On the theory I have advanced, the arrest of impressions from the afferent muscular nerves abolishes the muscle-reflex action on which the local irritability depends. Many other facts of disease show that this irritability is easily lost, and that a very slight change in any part of the reflex arc, too slight to cause other symptoms, is sufficient to arrest the knee-jerk.* Hence we can understand that this loss should be constant and early. It is not improbable, moreover, that these nerves suffer in special degree. The muscles may be insensitive to pain, *e.g.* that of electrical stimulation. Pressure and extension, which in health are painful, sometimes cause no sensation, even when the skin is sensitive.

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* In disease of the nerve-roots of the cauda equina there is always ascending degeneration of the posterior median columns, but not of the antero-lateral ascending tract. Hence the root-fibres from which the path is continued by this tract must end in nerve-cells in the posterior cornua, and the degeneration of this tract

The mechanism of the muscular inco-ordination, which is the obtrusive symptom of the disease, has been the subject of much discussion. Two fundamental facts, however, limit the problem. First, the ataxy cannot be primarily due to the loss of cutaneous sensibility. Disease of the conducting path in the cord may cause absolute anæsthesia of the skin without the least ataxy. Although this does not prove that interruption of the sensory path in the nerves, between the skin and the reflex centres, may not cause inco-ordination, this element seems to be excluded by the fact that there is no relation between the ataxy and the loss of feeling in the skin. There may be, in tabes, much ataxy without any cutaneous anæsthesia, and *vice versâ*. The second fact is that ataxy may exist in considerable degree when the lesion is solely one of the peripheral nerves, and the posterior columns of the cord are free from disease.† These two facts, taken together, seem to show that the ataxy may be produced by one

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in tabes is proof of the degeneration of these sensory cells. See the 'Lancet,' June 19, 1886.

* *E.g.* its loss after diphtheria when there are no other symptoms.

† Apart from the evidence of this from cases of true tabes, a very instructive case has been recorded by Dr. Hughes Bennett, in which all the symptoms of tabes were present in a case of multiple tumours of the posterior nerve-roots ('Clinical Soc. Transactions,' vol. xviii). Ataxy has also resulted from other processes, such as injury, that have caused extensive damage to the posterior roots alone.

mechanism, even operating alone, the disease of the afferent muscle-nerves. If the loss of the knee-jerk in tabes is to be taken, as I believe it may, as an indication of the disease of these nerves, the constancy of the loss shows the constancy of the presence of this element, in some degree. As just stated, a very slight degree of disease may abolish the knee-jerk; it is probable that a greater degree is needed to cause inco-ordination, and a still greater change to produce actual insensibility to pressure or extension.

In unilateral lesions of the spinal cord, as we have already seen (p. 229), there may be loss of the sense of posture, with intact cutaneous sensibility, on the side of the lesion, and no loss of this sense on the opposite side, on which cutaneous sensibility is lost. In such a case marked ataxy has been observed on the side on which the sense of posture was lost, when motor power returned.* Whatever effect in causing ataxy is produced by disease of the path of muscular sensibility in the spinal cord must also be produced by interruption of the path between the muscles and the cord. The latter must also arrest whatever reflex action depends upon these muscle-nerves,† and it is possible that such reflex action takes some part in the mechanism of muscular co-ordination.

In this connection it is important to note that the fibres that pass up by the posterior median columns probably constitute the path from these nerves. The root-fibres to the columns seem to pass up, as the path is proved to do, without decussating, and they may be diseased in intense degree when there is no loss of cutaneous sensibility.‡ This tract is affected in almost all cases of tabes in which there is a lesion in the cord, and in all such cases in which there is ataxy. This fact suggests that the lesion of the root-fibres is first and chiefly of those from the muscles.§

The posterior median columns (extensively connected with the cerebellum) and the direct cerebellar tract probably conduct to the cerebellum the impressions from the sensory muscle-nerves. If so, the disease of these tracts and nerves must interfere with the co-ordinating action of the cerebellum, and cause some part of the loss of function which results from disease of the cerebellum itself.

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* Gilbert; see foot-note, p. 229.

† In a case in which the inco-ordination was much greater in the right leg than in the left, and cutaneous sensibility was equally impaired in the two, a strong traction on the calf muscles, by forcible passive flexion of the foot, produced a distinct sensation in the extended muscles of the left leg, and no sensation in those of the right. In another case, in which the skin was sensitive to the slightest touch, the patient was unconscious of a vigorous extension of the toes produced by faradic stimulation of their short extensor.

‡ This posterior tract is most diseased on the side opposite to that on which there is most degeneration of the antero-lateral ascending tract, which probably conducts sensations of pain.

§ The opinion that the ataxy depends on impairment of the muscular sense was put forward in 1855 by Dr. Russell Reynolds; see p. 398. Déjérine has also associated the ataxy with the degeneration of the peripheral sensory muscle-nerves ('Archives de Physiologie,' 3rd series, vol. iii, p. 231).

There is another mechanism which may co-operate in increasing ataxy. The vertical fibres of the postero-external column have only a short course, and some probably connect the posterior grey matter at adjacent levels. The affection of these fibres seems to be proved by the fact that the comma-shaped bundle of fibres in the front of the post.-external column may be specially degenerated. This lesion may impair the association of the sensory structures, and so cause a want of harmony in the central mechanism. The theory that the ataxy was due to this cause was advanced by Todd in 1847. It is not probable that, if effective, it has more than an intensifying influence, since ataxy may exist when the posterior columns are throughout unaffected. The discovery that ataxy may result from alcoholic peripheral neuritis confirms the view of its origin above stated.

Although cutaneous anæsthesia cannot, alone, produce ataxy, it may reasonably be assumed to increase that which already exists. Sensations from the skin furnish important guidance to the motor cerebral centres, and are probably also concerned in such reflex muscular actions as that of standing. Similar guidance to the cerebral co-ordinating centres is also afforded by visual impressions, the loss of which distinctly augments the defect of co-ordination. The varying characters of the ataxy in different cases probably depend on the local variations in the degree of change in the muscle-nerves. The special effect of the disease of other elements of the cord, related to the posterior roots (as the posterior vesicular column), has still to be ascertained. The degeneration of the antero-lateral ascending tract is perhaps connected with loss of sensibility to pain.

It is probable that co-ordination is chiefly an automatic process, depending partly on muscle-reflex actions, and on the connection of neighbouring sensory structures in the spinal cord, but chiefly on the function of the cerebellum, determined by the connection of the muscles with it, and that the interruption of this connection is the chief element in the inco-ordination of locomotor ataxy. It is probable also that the automatic processes are in part under cerebral control, guided by sensory impressions which do enter the sphere of consciousness, and that the derangement of this control will intensify inco-ordination, though incapable of producing it. We do not at present know to what extent, in any given case, the symptoms are due to the cord disease or to the peripheral nerve lesions. Apparently pains, ataxy, and anæsthesia may be due to either. The question can only be decided by the comparison of symptoms and pathological changes in a large number of cases. That anæsthesia may be due to the peripheral changes is proved by the observed correspondence of the two in distribution (Déjérine).

The trophic changes in the skin, bones, and joints are probably due to the process of degeneration in the peripheral nerves. The degeneration has been found in all cases of the kind in which it has been looked for, and found also in the nerves of diseased joints. The pathology of the muscular wasting has been already mentioned.

The pains in the region of the fifth nerve are explained by the lesions in its root, especially in its ascending root, which, coming up from the medulla, is homologous with the posterior spinal roots. This nerve, indeed, as Pierret has pointed out, represents the sensory roots of almost all the motor cranial nerves. The degeneration of the optic nerve is fairly comparable with that of the peripheral spinal nerves. Considering the special character of the optic nerve, the absence of degeneration of the retina does not seem to destroy the analogy between the two. Regarding the pathology of the visceral crises we know but little. The changes observed by Pierret in the neighbourhood of the centre for the pneumogastric may be, as Buzzard has suggested, an indication of the cause of the disturbances that occur within the range of this nerve, especially of the gastric and laryngeal crises.

The transient motor symptoms in the limbs, and in the eyeball muscles, are apparently of functional origin. They must be distinguished from the lasting palsies, which are probably due to degenerative processes in the nerves or nuclei; the loss of function of the internal ocular muscles, being persistent, must be ascribed to degeneration of the related centres. But even the light-reflex may return after it has been absent for years, and its loss must therefore depend, at least sometimes, on molecular changes capable of recovery. It is indeed important to remember that tabes is a disease which begins as a derangement of molecular nutrition, which is probably only to be discerned when relatively great in its degree. If we also remember that this derangement is the result of some toxic material or virus circulating in the blood, we can understand better the leading facts of the disease, and especially the variations in its course, its degree, and the precise character of the symptoms.

Diagnosis.—The diagnosis of tabes rests on the combination of symptoms already described. In the early stage, the loss of the knee-jerk, together with pains, or unsteadiness on standing with the feet together and the eyes closed, justifies a diagnosis of the commencing affection, provided we can exclude toxic peripheral neuritis (especially that due to alcohol), diabetes, diphtheritic palsy, and also a lesion of the anterior cornua or nerve-roots by the absence of wasting of the muscles and change in their irritability. The diagnostic value of the loss of the knee-jerk can hardly be overrated. It is probably never absent in health. If there is doubt as to its loss, the precautions recommended on p. 15 should be adopted. When it is lost, and reflex action is in excess, a true reflex movement may sometimes simulate the jerk; the distinction is that the tap sometimes causes a movement and sometimes does not; that an interval, brief but appreciable, elapses before the movement occurs; that a similar movement is caused by a prick on the skin, and that the delayed contraction sometimes occurs in the other leg, or in the adductors of both legs.* In cases in which the knee-jerk is present, the diagnosis of tabes is only justified by distinct and characteristic inco-ordination. In the few cases of this kind that have come under my own observation, the knee-jerk has been either unequal on the two sides, or has been lost on one. Such abnormality is probably the rule in these cases, and the diagnosis is thus facilitated. In a case with lightning pains, but neither inco-ordination nor loss of knee-jerk, a suspicion of tabes would be justified by the presence of some other symptom, such as retention or incontinence of urine, loss of sexual power, or loss of the iris-reflex. But, as we have seen, it is probable that cases occur in which these pains exist alone,—a condition that may be termed "tabetic neuralgia."

The loss of the iris-reflex is of great diagnostic importance. It shows that a degenerative process is at work in the nervous system, and it suggests, therefore, that other symptoms are also due to degeneration. But since the iris-reflex is not always lost in tabes, the negative significance of a normal reflex is far less than is the positive significance of its loss. The practical value of this symptom can hardly be overrated. It puts the observer, so to speak, on the track of nerve degeneration. Its loss is, moreover, so often due to preceding syphilis, that it should always suggest this antecedent.

There are certain diseases with which tabes is especially liable to be confounded. One of these is multiple alcoholic neuritis. The ordinary form of this disease is readily distinguished by the symmetrical paralysis which is its chief manifestation. But the variety which closely resembles locomotor ataxy is the "alcoholic pseudo-tabes" (p. 125), and in this the diagnosis may be very difficult. This is not surprising, because it resembles tabes pathologically; the lesion is a "parenchymatous neuritis," a subacute degeneration, beginning in the nerve-fibres, and similar to the peripheral form of tabes. It often affects, however, in some degree, the motor as well as the sensory nerves, and there is then some weakness in the distal portions of the limbs. An altered electrical reaction may be found in the muscles (never in the weakness sometimes met with in early tabes), and there is generally marked muscular tenderness, scarcely ever present in tabes. The pains have not the "lightning" character, and the sphincters escape. The pupils act normally, but this is not an absolute distinction, since they may be unaffected in tabes. A history of alcoholism may help the diagnosis, and so may improvement when alcohol is withdrawn. By attention to all these points, a diagnosis can generally be made without much difficulty.

When the symptoms and lesion of tabes are combined with those of general paralysis of the insane, it may be doubtful in which category a case should be placed. The question is rather one of the preponderance of the symptoms of one or the other malady than of absolute distinction between them. In most cases, however, in which this combination exists, the symptoms of general paralysis become more pronounced as time goes on, and the spinal symptoms, which at first were the most conspicuous, pass into the background.

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* It is impossible to exaggerate the difficulty presented by some patients in ascertaining whether the knee-jerk is or is not lost, and the care needed, and repeated observations that are desirable, in doubtful cases.

All common forms of paraplegia are distinguished by the early loss of power, and by the persistence of the knee-jerk, often emphasized by its excess and by the occurrence of a clonus in the rectus or the calf muscles. If weakness supervenes in tabes, it is usually late in the course of the disease, and the other symptoms have been and are well marked. If the knee-jerk has been once lost in this disease, it is not reproduced by secondary lesions of the cord which ordinarily increase the knee-jerk. The same distinctions usually suffice for the diagnosis from the combined form of paralysis and inco-ordination that I have termed "ataxic paraplegia." In this the knee-jerk is excessive, and there is foot-clonus, while pains and anæsthesia are absent; spasm supersedes ataxy. In other forms of combined disease of the cord the diagnosis depends chiefly on the recognition of union.

Acute lesions of the cord, probably situated in the postero-external column, may produce inco-ordination and pain, but the suddenness of the onset, the limitation of the symptoms, and their tendency to subside, usually suffice for the distinction. Symptoms from this cause are far more common in the arms than in the legs. Ataxy in one limb may also be produced by a tumour growing in the posterior column, but other symptoms indicate the invasion of adjacent structures, as was conspicuous in the case figured in the chapter on tumours of the cord. All these cases present the characteristics of a focal lesion with random consequences, rather than of a system disease with limitation to a special function.

In diphtheritic paralysis occurring long after the sore throat, or in which the nature of this was not recognised, the loss of the knee-jerk may cause tabes to be suspected; but the nature of the case is almost always rendered clear by the paralysis of accommodation and of the palate which precedes the ataxy, and should be suggested by the absence of pains and the presence of weakness. If true tabes follows diphtheria, the sequence is too rare to be of practical importance.

When the dorsal region of the cord is chiefly affected, the severity of the pains in the trunk may lead to a suspicion of disease of the vertebral bones, but in the latter the pain is local and fixed, and this, with the characteristic increase of pain when the patient moves, should prevent error. It is still more frequent to mistake such cases for neuralgia of the intercostal nerves, but even if there are no pains in the legs to suggest tabes, the knee-jerk and the light-reflex of the iris are lost in tabes. Experience shows that the most frequent errors in diagnosis are due to a mistake as to the nature of the pains, which are mistaken for rheumatism, especially when influenced by weather, or for sciatica when referred to the region of the sciatic nerve, or for some form of gout if the disease exists in the patient or his family, or in the mind of the physician.*

The only organic intra-cranial disease that may be mistaken for tabes is tumour of the cerebellum. In some cases of tabes the unsteadiness resembles that of cerebellar origin. Loss of the knee-jerk is strongly in favour of tabes; in cerebellar tumour, increase of the jerk is the rule, and loss is only to be regarded as not *absolutely* incompatible. Anæsthesia and lightning pains are conclusive evidence of the spinal disorder, while in cerebellar tumour severe occipital headache is almost invariable, and considerable optic neuritis is extremely common. Optic nerve atrophy is primary in tabes, post-neuritic in tumour.

The various visceral crises are often mistaken for primary disorders of the deranged organs. Recurring attacks of the character described should always arouse suspicion of their possibly tabetic nature, and an examination of the knee-jerk will usually decide the point.

PROGNOSIS.—The prognosis in tabes corresponds with the facts stated as to the course of the disease. It does not, as was once thought, deserve the epithet "progressive," given to it before the pre-ataxic stage was known and before the disease had been widely observed. Arrest is frequent, and considerable improvement is not rare. The earlier the stage of the disease, the better is the prognosis. In the first stage, the disease often becomes stationary. When ataxy is developed, the prospect of arrest is less than in the first stage, but is still considerable. Occasionally, even in the typical form of the disease, the amount of improvement is very great. One patient was scarcely able to walk across the room, in consequence of the inco-ordination, when he came under treatment, and at the end of six months his gait was scarcely distinguishable from that of a healthy person. When the third stage is reached the possibility of improvement is far less, but is not quite absent. A woman suffering from pure tabes, when admitted to hospital, had been unable even to stand for six months, on account of the extreme ataxy; but she improved so much that in a few months she was able to walk about the room without help, and has continued, now for six years, to do her household work without difficulty, and even to walk some miles with the aid of a stick. On the other hand, in some cases the malady increases in spite of every effort, and even when there is permanent arrest or improvement it is rare for all symptoms to disappear. The knee-jerk generally remains absent, and often attacks of pain continue to occur. It is difficult to find any trustworthy indications to guide the prognosis in an individual case. The fact of preceding syphilis has little influence on the prognosis.

* In one case, in which tabetic trunk-pain was chiefly felt at the level of the epigastrium, a physician of repute not only made a diagnosis of gastric gout, but refused to modify his opinion when loss of the knee-jerk was discovered, and the patient became unsteady in gait.

Only when the symptoms have developed rapidly and the syphilis is recent is there some ground to anticipate good from antisyphilitic treatment. In all cases, however, in which the onset of symptoms is acute there is more prospect that they will pass away than if they develop slowly. In most cases the only trustworthy guide to prognosis is the observed tendency of the individual case.

Of special symptoms, the pains are often the most distressing, and unfortunately they are the most obstinate symptom of the disease. They may persist in undiminished severity when other symptoms lessen; and, on the other hand, they may become trifling when the disease increases. Diminution of the pain is a good sign if other symptoms are stationary, but not if these increase. It is often, however, a solace to the patient to know that persistence of the pains do not mean progress in the disease. The optic nerve atrophy is usually progressive, but not invariably, as is often asserted. In a few cases I have known its progress to be permanently arrested. Strange to say, the occurrence of optic nerve atrophy makes the prognosis as to the spinal symptoms better; in most cases of atrophy the spinal lesion remains in the first stage.

TREATMENT.—The disease is one in which treatment often has a very clear influence, not only in relieving suffering, but in determining the arrest or diminution of the disease. This is true especially of treatment by drugs. But the first care should be to secure freedom, as far as possible, from all influences capable of causing the disease, and from whatever is likely to depress the nervous system. Excessive mental work, anxiety, and physical fatigue are all harmful. In severe and acute cases, absolute rest for a week or two is often useful at the onset of treatment. If the patient walks about, exercise should always stop short of fatigue. Care should be taken to avoid the risk of falls; a severe concussion of the spine will sometimes excite to fresh activity a stationary disease. Exposure to cold is also injurious; a severe exposure may cause acute mischief. If practicable it is well that the winter should be spent in a warm and dry climate. A sea voyage is sometimes useful, combining, as it does, a maximum of fresh air with a minimum of exertion; it has, however, less actual effect than might be expected. If there is much ataxy, the risk of falls from the motion of the ship must be taken into consideration. It is the most useful in cases that are already stationary; such patients often return with a diminution in all their symptoms. When the disease is advancing, a voyage often does harm rather than good.

The digestive organs should be kept in careful order; an attack of indigestion or constipation is often attended by lightning pains, which cease when an aperient has acted. Food should therefore be light in quality, and easily digestible. The change from an active to an inactive life, which tabes often causes, may lead to the development of gout in an individual predisposed to it, either by inheritance or by his previous mode of life; and the tendency must, as far as possible, be obviated by regulation of diet and by frequent aperients. Smoking should be either avoided or limited to a small amount. Alcoholic excess is especially harmful; it sometimes excites a very severe exacerbation of the symptoms; this is not surprising, since, as we have seen, chronic alcoholism may cause a disease resembling tabes in its symptoms and pathology. Sexual excess is also most injurious. Many patients are preserved from this danger by the disease itself, but it is not always so. Excess seems to have a special influence on the optic nerve atrophy. Several very painful instances of this have come under my notice. A man in the early stage of tabes, with slight atrophy, some peripheral limitation of the fields, but little impairment of acuity of vision, started on a voyage from Australia to England. The day before he started he married, and when he reached this country he was quite blind.

The influence of drugs on the disease is unquestionable in many cases, and if they are used wisely, the more a physician sees of the disease, the more highly is he likely to esteem them. There is no specific for the malady, and in some cases the morbid tendency over-powers every influence that can be brought to bear upon it. Moreover, the influence of drugs is variable; that which seems to do much good in one case has no influence on another, and this is true also of the same case at different periods. Hence the therapeutic statements that have been made by various observers are somewhat discordant.

The first question is the influence of antisyphilitic agents. In tabes such influence is distinct only in rare cases, chiefly under the conditions mentioned in the section on prognosis. In those circumstances full doses of iodide of potassium should be given, and if the interval since the primary disease is short, mercury may be rubbed in until the gums are slightly affected. In cases of slow development, several years after primary syphilis such treatment seldom does good, and sometimes does harm. But so widely spread is the opinion that every consequence of syphilis is amenable to treatment, that it is often well to avoid the suspicion of neglect, and even to clear the therapeutic ground, by a course of iodide of potassium for five or six weeks. Very small doses of mercury, such as $\frac{1}{16}$ grain of the red iodide, may also be given for a longer time, and may often be conveniently combined with other drugs. It seems sometimes to have a tonic influence, and perhaps may lessen the secondary subinflammatory process in the cord and pia mater, which, as we have seen, sometimes constitutes part of the morbid process. But in such late cases, and in all the late nerve degenerations that follow syphilis, energetic mercurial treatment only does harm. Its depressing influence often increases the degenerative tendency, and hastens the progress of the disease.

Of drugs that have been recommended for tabes, those that are most useful are arsenic, iron, quinine, aluminium, nux vomica and strychnia, nitrate of silver, Calabar bean, belladonna, ergot, phosphorus. Of these arsenic is certainly that which most frequently does distinct good. Most of the cases in which I have known the greatest improvement to occur, have been taking it at the time. In several of these cases there was no other change in the conditions of life to which the improvement could be ascribed. The form in which arsenic is given matters little; ♏v or ♏vij of the Liq. Arsenicalis may be given in a mixture, or $\frac{1}{12}$ gr. of arseniate of soda in a pill. Iodide of arsenic has no special advantage, and is not so well borne. This or any other drug must be continued for at least six weeks before an opinion of its influence can be formed. Rapid improvement can never be expected in so chronic a disease. But it is not well to continue any drug for more than about three months at a time. Most good is effected by alternating two or more; the return to one that had ceased to do good is often beneficial. In connection with the influence of arsenic, it is interesting to note the well-known effect of the drug in improving the nutrition of the skin, and the frequency with which the cutaneous nerves are degenerated in tabes. Quinine, or the extract of nux vomica, may be usefully combined with arsenic; or a small dose of mercury may be also given, ♏xv of Liq. Hyd. Bichlor. with ♏v of Liq. Arsenicalis, or $\frac{1}{24}$ gr. of the red iodide with $\frac{1}{12}$ gr. of arseniate of soda in a pill. Occasionally arsenic seems to irritate the nerve-centres, but a very small dose may then be borne with advantage. Nitrate of silver and phosphorus are less useful, but may sometimes be given alternately with arsenic. The chloride of aluminium is another agent that I have found of distinct service, 2—4 gr. twice or three times a day.

Calabar bean has been recommended by Ringer as producing temporary improvement in many chronic spinal diseases. Its influence in tabes is not great. Ergot has been esteemed highly by some, but is seldom distinctly effective; it is most likely to be useful if the symptoms suggest that there is a secondary subinflammatory process. Belladonna is sometimes useful, and strychnia often produces marked improvement, especially combined with a small dose ($\frac{1}{100}$ gr.) of nitroglycerine, which causes the blood containing the tonic to pass more freely to the nerve-centres.

Counter-irritation to the spine, by blisters or the actual cautery, is sometimes useful, chiefly in the cases just mentioned, in which the disease develops, or the symptoms increase, rapidly, or in which there is spinal pain or tenderness. It is not probable that it influences the process of degeneration, but it may lessen any secondary inflammation.

Of special symptoms that require treatment, the pains are the most important. Unfortunately they are often very obstinate, and their variability renders it difficult to estimate the influence of treatment upon them. Severe paroxysms may yield only to hypodermic injections of morphia, but this should be reserved for the most intense attacks, since its use is undesirable for a recurring symptom which may continue for years. Indian hemp is often effective; gr. ¼ or ½ should be given every three or four hours during an attack. But the most useful agents for relieving a paroxysm are antipyrine and acetanilide. One or the other of these seldom fails; should they do so, exalgin or phenacetin may succeed. Hypodermic injections of cocaine generally arrest, for several hours, pains that are superficial in character, especially if accompanied by hyperæsthesia of the skin. The injection should be of $\frac{1}{6}$ to $\frac{1}{4}$ of a grain, at the upper part of the region to which the pain is referred. Cocaine has no influence over the deeper pains. Superficial pain is also relieved by the local application of chloroform sprinkled on spongio-piline (or lint and oiled silk). Occasionally the pains are lessened by a warm bath. Counter-irritation to the spine, as by repeated sinapisms, may give some relief. These measures, however, only relieve paroxysms, and do not prevent recurrence. This is sometimes effected by a course of chloride of aluminium.

Paroxysmal visceral symptoms often yield to antipyrine or acetanilide, but when severe, morphia alone mitigates their intensity. Slight gastric crises may be relieved by simultaneous sinapisms to the epigastrium and neck. Laryngeal spasm is usually at once relieved by nitrite of amyl or nitro-glycerine; the local application of cocaine is also useful. Weakness of the bladder is sometimes lessened by nux vomica or strychnia. The latter may be given by hypodermic injection, as recommended in the chapter on muscular atrophy. Nocturnal passage of urine is occasionally arrested by belladonna.

Blisters or sores on the feet should receive careful local treatment until they are well. If neglected they may become very troublesome. The patients should be cautioned never to cut a corn; a perforating ulcer is often set up by a corn being cut too deeply. The epidermis should be softened with an alkali and rubbed off with pumice-stone.

Attention to the bladder is one of the most important elements in the treatment of tabes, as in that of all diseases of the spinal cord, chronic and acute. If there is any reason to believe that the bladder is imperfectly emptied, a catheter should be passed; and if residual urine is found, the bladder should be emptied perfectly and washed out every day or every other day. No hesitation need be felt in the use of the catheter in these cases. Many patients die every year from want of the catheter, because it is thought that all is right since they pass urine freely. The residual accumulation, or the slight cystitis, sets up chronic pyelo-nephritis, which develops insidiously, and is unsuspected until mysterious febrile disturbance occurs, chronic or acute, and leads to death. It is probable that the onset of the final acute disturbance is sometimes determined by the passage of a catheter, just as this will cause a rigor or transient hæmaturia in a healthy person. I have seen many melancholy instances of death from the consequences of unsuspected retention, and I have never seen any case in which the early and frequent use of the catheter did harm. It is especially important to insist on the danger that the practitioner may be misled by the patient, who thinks that because he passes urine freely he necessarily empties his bladder perfectly.

Electricity has little influence on the chief symptoms of tabes. The voltaic current is powerless over either the pains or the ataxy, whether it be applied to the spine or the limbs. Faradisation of the skin by the wire brush has been recommended for the defect of cutaneous sensibility, but it has little if any influence. Temporary improvement in gait, and sometimes a little permanent improvement, may be obtained by faradisation of the muscles; the stimulation of the afferent muscle-nerves is probably the mechanism of its action. When the bladder or its sphincter is weak, faradisation from the hypogastrium to the perineum may be employed, and occasionally seems to do some good. In secondary wasting of the muscles an attempt may be made to maintain their nutrition by stimulating them with whatever current they respond to, but the central cause usually renders local treatment ineffective.

Nerve-stretching was fashionable a few years ago, the operation being usually performed on the sciatic nerve. In the first case in which it was employed (by Langenbuch) the procedure was followed by remarkable and mysterious improvement, not only in the pains, but also in the inco-ordination. The patient subsequently died under chloroform, administered in order that the nerves of the arms might be stretched. The spinal cord was found by Westphal to be healthy. Although the nerves were not examined, there can be little doubt that it was one of the cases in which the nerves alone are diseased. The operation has since been performed in a large number of cases, but without results that have secured its survival, and it is passing into merited disuse. Its place has been taken by extension of the spinal column. In this procedure the patient is suspended in such a manner as to allow the weight of the body to rest on the head and shoulders, and head alone, alternately, and the ligaments of the spinal column are supposed to be so stretched that the spinal cord and its nerve-roots are elongated. The chief effect is produced on the ligaments of the upper part of the spine, but the actual amount of elongation of the spine is exceedingly slight and insignificant, as has been proved by experiments on the dead body. It is difficult to conceive any mechanism by which it can do the good in tabes which was at first ascribed to it; and the careful observations by Russell and Taylor on a large number of cases at the Queen Square Hospital* seem to show conclusively that it has no real power of influencing the disease. It will also probably before long be forgotten.

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* Russell and Taylor, 'Brain,' Summer Number, 1890, vol. xiii, p. 206.

PRIMARY SPASTIC PARAPLEGIA

(PRIMARY LATERAL SCLEROSIS).

The morbid state thus designated is one that has been, and still is, the subject of much discussion. Its relations are complex, and a somewhat lengthy pathological introduction is necessary.

General Pathology.—We have seen that in every kind of transverse lesion of the spinal cord, provided this is situated above the lumbar enlargement, the paralysis of the legs is soon accompanied by excess of myotatic irritability (increased knee-jerk, foot-clonus), and that the muscle-reflex action, on which this irritability (and muscular tone) seems to depend, gradually increases to tonic spasm, so that a condition develops to which the term "spastic paraplegia" is applied. A condition quite similar to that which is thus secondary to a transverse lesion often develops gradually, without any indication of a primary focal disease, and without any sensory symptoms to indicate that the mischief extends beyond the purely motor elements of the cord. We have also seen that these symptoms indicate disease of the upper segment of the motor path, the cortico-spinal segment (p. 176), which extends from the motor cortex through the pyramidal tracts, and ends in the grey matter of the cord, doubtless by a subdivision and ramification of the nerve-fibres in the fibrillary network of the spongy substance. From the gradual onset and limitation of the symptoms in these cases, it has been assumed that the disease consists of a primary sclerosis of the pyramidal tracts, i. e. in a degeneration of the fibres of this upper segment. Since these tracts run chiefly in the lateral columns, the disease has been termed "lateral sclerosis." The clinical features presented by these cases, and their probable significance, were first pointed out by Erb.* Degeneration of the pyramidal tracts had been already observed, by Türck (1856) and Charcot (1865). Erb's inference as to the nature of these cases was supported by the independent (and indeed previous) researches of Charcot on cases of muscular atrophy; he showed that in such cases muscular rigidity coincides with degeneration of the pyramidal tracts. Pathologists have since been searching for confirmation of the hypothesis—for evidence that the symptoms, in their pure form, without muscular atrophy, depend on degeneration limited to the pyramidal tracts. Such degeneration in slight degree, associated with slight symptoms, has been found in cases of general paralysis of the insane (by Westphal and others); but in all other cases that have been examined, either other parts of the white substance have been degenerated, or the disease has involved also the anterior cornua.†

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* In 1875, 'Berlin klin. Wochenschr.,' No. 26; 1877, 'Virchow's Archiv,' Bd. lxx.

† In a case recorded by Stofella no other part than the pyramidal tracts could

In some cases, as one published by Dreschfeld (of which a figure is given at page 447), the change in the anterior cornua has been so slight that the required conditions are nearly fulfilled.

Although absolute demonstration has not yet been furnished, the indirect evidence of the correctness of the pathological hypothesis is very strong, and the only open question is whether, when there is no disease of the motor nerve-cells of the anterior cornu, the degeneration is so limited to the pyramidal tracts as to constitute a system disease in the strict sense of the word. It is not surprising that demonstration of the nature of the pure cases is not forthcoming, since, as we shall see, the disease has little tendency to shorten life.

It should, moreover, be noted that cases which present this group of symptoms may be very various in nature. We have seen (p. 212) that, in each segment of the motor path, the same symptoms are produced by disease of any part of the segment. In the upper segment, with which we are now concerned, the symptoms are the same, whether the disease is in the cortex of the brain, the internal capsule, the pyramids of the medulla, or the pyramidal tracts of the cord; and they must also be the same if the disease is limited to the termination of the segment in the grey matter of the cord. In cerebral hemiplegia the state of the arm closely resembles that present when primary spastic paraplegia involves this limb. The leg in hemiplegia presents a less close resemblance to its condition in spinal disease, because the leg is innervated from both cerebral hemispheres, and the supplementary influence of the hemisphere of the same side lessens the effect of the disease of the hemisphere of the opposite side. But if there is disease of the leg-centres in both hemispheres, the state of the legs may be identical with that resulting from disease of the spinal cord. Such bilateral disease often results from injury during birth,—meningeal hæmorrhage over the upper part of the central convolutions. This resulting condition is termed "congenital spastic paraplegia." (See vol. ii.)

On the other hand, it is extremely probable that degeneration begins in the termination of the fibres in the grey matter, just as it does in the extremities of the peripheral nerves. It may even be limited to these terminal parts, as the extremity of the lower segment is paralysed alone by many agents. In such disease the white columns would be found normal, as in one recorded case in which the symptoms of spastic paraplegia existed during life, and no anatomical change was discovered after death. The detection of the disease of the terminal portion in the grey matter is extremely difficult, perhaps impossible, because the structure must consist of nerve-fibrillæ that interlace with others that are unaffected. This view is supported by the fact that in other cases some degeneration has been found in the lateral pyra-

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be seen diseased on naked-eye observation, but no microscopical examination was made.

midal tracts in the lumbar region, slighter than the intensity of the symptoms suggested, and gradually lessening, to cease higher up the cord. Such a condition is comparable to the degeneration of the lower portion of the second segment of the motor tract in some cases of peripheral (degenerative) neuritis, in which there is a degeneration of motor nerve-fibres, greatest in their extremities, extending for a variable distance up the nerves, but lessening long before the spine is reached. It is highly probable that the toxic agents which cause lathyrism and pellagra (*q. v.*) act on the extremities of the pyramidal fibres, as curara does on the nerves; among the spinal symptoms of lathyrism are paralysis of the legs with rigidity and contractures, and increase of myotatic irritability—foot-clonus, &c.*

Both clinical and pathological evidence shows that the morbid state often occurs also as part of a more extensive degeneration. Degeneration may occur in both lateral and posterior columns, giving rise to combined paralysis and ataxy—"ataxic paraplegia," sufficiently definite in its clinical characters and course to make its distinction convenient; it is therefore separately described. The following account of the clinical features of spastic paraplegia is founded on cases in which the motor paralysis and spasm existed alone, with no definite sensory loss, in which they came on gradually, with nothing in the state of the patient or the history of his symptoms to suggest either a focal lesion or an acute process.

CAUSES.—An inherited neuropathic tendency is to be traced occasionally in this as in other chronic spinal diseases. Thus in one case there was a history of insanity in uncle, aunt, and two cousins. The disease affects both sexes in almost equal frequency, presenting in this a contrast to posterior sclerosis. The period of life at which it most frequently begins is between twenty and forty; about three quarters of the cases begin in these two decades, and about an equal number in each. Cases frequently commence, however, in the second decade of life, chiefly in its latter portion. After forty they become much less common; the latest age at which I have known a typical case to commence is sixty-one.

The disease sometimes follows syphilis in a way to suggest a causal relation, even when all cases are excluded in which there is any reason to suspect a focal lesion. In one case the symptoms commenced six months after the primary disease.† But this antecedent is not frequent, and in this respect also the disease contrasts with posterior sclerosis. Proximate causes are to be traced only in a minority of the cases. The most frequent is concussion of the spine, such as a fall on the back. Some time, often two or three years, elapses between the fall and the first pronounced symptoms of the disease, and hence focal lesions due to the fall can be excluded. Next in frequency is repeated exposure to wet cold. Very rarely the symptoms have slowly followed some acute illness. In several cases the disease has succeeded prostration after childbirth or abortion, or has commenced during lactation. In one case an attack of arthritis in the knee and ankle of one leg was the immediate antecedent, and this leg was the first to become weak. In another case the symptoms followed subacute arthritis of both knee-joints, apparently rheumatic in nature. The possibility that a joint inflammation may be of spinal origin must be borne in mind in considering the significance of such cases, but it is on the whole probable that a primary joint affection is an occasional cause of the spinal disease.* (Acute arthritis, certainly of spinal origin, has only been observed in severe myelitis.) The cause of the congenital form is always injury to the brain during birth, in most cases meningeal hæmorrhage, causing compression of the motor cortex.

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* See Marie, 'Prog. Méd.,' 1883, No. 43.

† The cord lesion was found to be sclerosis, but the direct cerebellar tract was also degenerated (Minkowski).

SYMPTOMS.—Weakness of the legs, of very gradual development, is the first symptom. The patient finds that he gets tired more readily than before, and that the legs feel heavy; sometimes one leg becomes weak before the other. The progress of the weakness is very variable, but it is slow in all characteristic cases. In many instances the patient is still able to walk a mile or two, even after the disease has lasted for several years, slowly increasing. On the other hand, walking power may be almost lost at the end of six months. It is doubtful whether more acute cases belong to this category. The early weakness is often accompanied by slight unsteadiness, chiefly subjective. When the patient seeks advice, it will generally be found that there is very distinct loss of power in the flexors, often greatest in the flexors of the hip, but considerable also in those of the knee and ankle, and occasionally much greater in the last than elsewhere. The knee-jerk is excessive and quick; the rectus contraction can be obtained, as the patient lies, by tapping the depressed patella, and a rectus-clonus is often obtainable by sudden depression of the patella. The foot-clonus is usually also obtained with readiness. In rare cases, in which the upper part of the legs suffers most, there may be a rectus-clonus, but only slight indications of a foot-clonus, two or three movements quickly ceasing. The tendency to spasm is at first noticeable as slight stiffness of the legs on first rising in the morning, but it gradually increases in degree as power lessens, until at last the legs, whenever extended, pass into a condition of strong extensor spasm, rigidly fixing them to the pelvis, so that, as the patient lies, if one leg is lifted from the couch by the observer, the other leg is moved also. The spasm may be such that the knee cannot be passively flexed by any force that can be applied to it until the spasm has become less. When flexed the limb is comparatively supple; but if it is then extended, the spasm instantly returns, making the limb rigid, and often completing the extension, just as the blade of a knife opens out under the influence of its spring, "clasp-knife rigidity."

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* See 'Arthritic Muscular Atrophy.'

The spasm is, roughly speaking, proportioned to the loss of power, and its extensor character may enable the patient to stand, the legs being fixed and rigid, when muscular power is quite insufficient to support the body when it is unaided by the spasm. In a still greater degree a voluntary effort may only excite general spasm of the limb instead of causing a definite movement. Occasionally there are also paroxysms of brief flexor spasm causing the legs to draw up. This occurs chiefly when the patient is in bed, and especially during sleep. When the spasm is great, a paroxysm may pass into violent clonic spasm, but this is merely the same clonus that can be produced by passive extension, excited by the tension of the spontaneous spasm (see p. 222). Reflex action from the skin is also usually excessive, and the stimulus excites an attack of spasm.

The gait of the sufferers from spastic paraplegia is very characteristic. The legs seem to drag behind the patient, and, in walking, each is hauled forwards as a rigid whole, the toes catching against the ground, and, when the ball of the foot rests on the ground, the limb may shake from the clonus developed by the extension of the calf muscles. When the patient sits, a similar trepidation occurs until the patient pushes the leg forwards so that the heel rests also on the floor. The muscles of the legs are usually large and well nourished. They often seem, indeed, to be hypertrophied, and perhaps are really so: the contraction of spasm constitutes a physiological stimulus to growth, just as does voluntary effort. But they are not always large; they are sometimes moderately wasted. The electric irritability is usually perfectly normal, but in the muscles that present the slight wasting just mentioned, there may be a trifling diminution of irritability, to faradism and voltaism alike. When the patient ceases, or almost ceases to walk, contracture sometimes occurs in the calf muscles. An active contracture is indeed common, preventing at first passive flexion of the ankle beyond a right angle; but if the pressure is maintained, the muscles yield and full flexion is possible. But in some cases there is the fixed contracture described on p. 214 (form 3). Flexor contraction at the hip or knee may occur also in the same cases under the influence of posture, but is on the whole rare, the tendency to it being counteracted by the extensor spasm.

The arms are often unaffected, but they suffer in some cases, and present the same progressive weakness and excess of myotatic irritability. Vigorous contractions are elicited by a tap on the tendon of a muscle, or on the bone to which the tendon is attached, provided the muscle is extended. A clonus can sometimes be obtained in the flexors of the fingers. There is less paroxysmal spasm than in the leg, but much rigidity of the limb, from active muscular contracture, flexing the elbow and flexing the fingers at all the joints, as in the "late rigidity" of hemiplegia. The difference in the form of spasm in the arm and leg is no doubt connected with existence of a reflex extensor mechanism, in the lumbar centres, concerned in the act of standing. The muscles of the arm are often as well nourished as those of the leg. The affection of the two arms is very rarely equal; it is common for one arm to be almost normal when the other is much affected and both legs are equally involved. Sometimes, however, the arm and leg on one side are paralysed, and the limbs on the other side are so slightly involved that, in comparison with the palsied members, they seem normal; the patient often believes that they are unaffected, but examination always reveals some weakness and an abnormal degree of myotatic irritability.

The muscles of the trunk may also suffer. A subjective "feeling of weakness" in the back is often complained of early, but seems to be as much a sensory as a motor symptom. Painful spasm of the abdominal muscles may occur in severe cases, and I have even known attacks of tetanoid rigidity of the back to be produced by attempts to move.

It is not common, in typical cases, for the symptoms to extend into the region of the cranial nerves, but, in rare instances, difficulty of swallowing and of articulation has existed, due to a similar palsy of the bulbar nerves. Excessive myotatic irritability has also been observed in the muscles of mastication, so that a tap on the chin causes a vigorous elevation of the depressed jaw (Beevor).

Sensory symptoms are often entirely absent, with the exception of slight dull pains in the legs, or more frequently in the back. The sensation of weakness in the back, already mentioned, occasionally reaches an intense degree. Rheumatoid pains may be troublesome, but sharp pain is rare, except in untypical cases to be presently mentioned. In such cases also there may be very slight defect of sensibility—such, for instance, as blunting of tactile sensibility on the finger-tips. Subjective sensations of "numbness," tingling, formication, are more common. They may exist for years without the development of any other anæsthesia, in spite of the progress of the motor symptoms.

The sphincters are sometimes affected, occasionally very early; often, however, they escape even to an advanced stage. Sexual power may be lost, or may remain, even when the spastic paraplegia has reached a high degree. The nutrition of the skin and joints undergoes no change.

Ocular symptoms are rare, with the exception of nystagmus, which will often be found if searched for. The action of the pupils is usually perfect, and I have only once seen optic nerve atrophy in an uncomplicated case.

The infantile form may resemble very closely that which occurs in adults. There are the same extensor spasm and increase of all forms of reflex action. As the child sits on the knee or a chair any sensory stimulus will make the legs shoot out in spasm (Fig. 125). But the rigidity does not reach the extreme degree often attained in the common form. The excess of the knee-jerk is always distinct, but a foot-clonus is not often to be obtained, perhaps because the muscle-reflex mechanism related to the calf muscles has not received the functional development that must result from the process of walking, with its

FIG. 125.

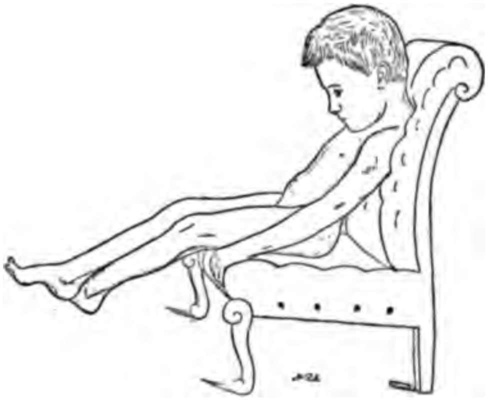

FIG. 126.

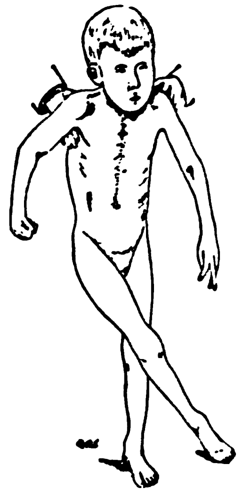

FIG. 125.—Congenital spastic paralysis (cerebral). Extensor spasm in the legs excited by a sensory impression.*

FIG. 126.—Infantile spastic paralysis of cerebral origin: cross-legged progression. (Drawn by Dr. Spencer, from a photograph.)

recurring sequence of tension and contraction. The active contracture in the calf muscles, which most cases present, is a serious hindrance to walking even when the muscular power is sufficient, and it is long before the attempt overcomes the contracture. In most cases, however, the child ultimately gains the power of walking, although much later than normal, and often with some peculiarity of gait, sometimes a tendency to "cross-legged progression," in which one foot gets over or in front of the other (Fig. 126), or with a swinging oscillation of the body, which may persist to adult life. The growth of the legs is often hindered.

The arms do not present tonic spasm such as is seen in adult cases. There may be a choreoid disorder of movement, spontaneous irregular movements with inco-ordination, but in the cases that can fairly be called "spastic paraplegia" the arm symptoms are slight. When considerable the condition is often termed "double athetosis." Its characters are described in vol. ii.

Transitional forms are met with which constitute gradations between primary spastic paraplegia and other degenerations of the spinal cord. Slight inco-ordination may co-exist in cases approximating the "ataxic paraplegia" described in the next section. Cases may begin as pure tabes, and indications of lateral sclerosis may be superadded. Slight muscular wasting in the arms may be associated with indications of lateral sclerosis in the legs, and constitute a transition to the form of spinal muscular atrophy to which the name "amyotrophic lateral sclerosis" has been given by Charcot. Very rarely considerable muscular wasting succeeds spasm in the same part.

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* Drawn by Dr. H. R. Spencer, from a photograph by Mr. Hyde Marriott.

Lateral sclerosis of the spinal cord (like posterior sclerosis) often forms part of the morbid process that underlies general paralysis of the insane. In some cases of this character the mental symptoms are extremely slight, and the case may have the aspect of a primary spinal disease, with slight mental failure complicating it.

The course of characteristic cases of primary spastic paraplegia is thus very chronic. The malady may, at any stage, cease to advance. Slight symptoms may remain stationary for twenty years. Often, however, arrest only occurs when the disease has reached a considerable degree. It is perhaps the least dangerous to life of any chronic spinal disease. Even secondary kidney trouble scarcely ever occurs; perhaps the excessive reflex action may save the bladder from injurious over-distension. It is when other elements of the cord suffer that dangerous complications ensue.

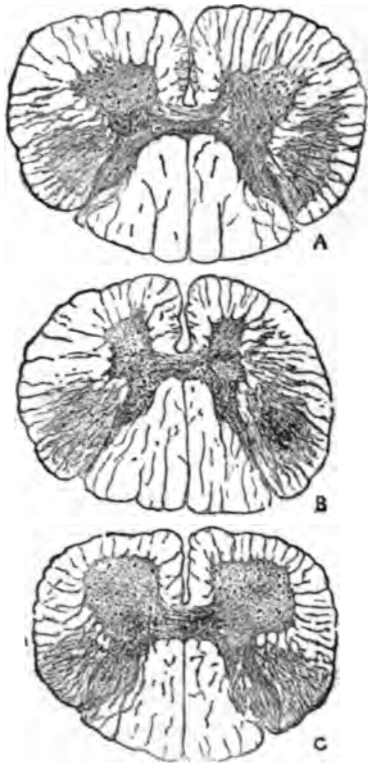

FIG. 127.—Sclerosis of the lateral and (in the cervical region anterior) pyramidal tracts, with slight degeneration of the anterior cornua. A, cervical; B, dorsal; C, lumbar sections.*

PATHOLOGICAL ANATOMY.—In the fact just stated we probably have an explanation of the silence of morbid anatomy on the subject of uncomplicated lateral sclerosis. "Nec silet mors," the apt motto of the Pathological Society, is true chiefly of disease that kills. Complete degeneration of the pyramidal tracts, anterior as well as lateral, is met with chiefly in cases in which the anterior ganglion-cells and motor nerves are also diseased, although in some instances the amount of this disease is small, and limited to the cervical region. An instance

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* I am indebted for these sections to Dr. Dreschfeld, who has published the case ('British Med. Journal,' Jan. 29th, 1881).

of such degeneration is shown in Fig. 127. The degeneration in the lumbar enlargement is usually limited to the lateral tract, but the anterior tract may be affected higher up the cord, as in 127, A. The lateral sclerosis is bounded externally, in the dorsal and cervical regions, by the normal direct cerebellar tract, and internally by the narrow "lateral limiting layer" which intervenes between the pyramidal tract and the intermediate grey matter. There is the usual increase of connective tissue and wasting of nerve-fibres. In many cases granule-cells are abundant in the affected area. They are always present in cases of short duration, and indicate the stage rather than the nature of the process. The degeneration probably begins in all cases in the nerve-elements themselves. In most cases many nerve-fibres can still be seen, scattered through the sclerosed area; most of them are fibres which lie in the pyramidal tract, but do not belong to it. The degeneration of the tract has been traced through the medulla, pons, and cerebral hemispheres to the motor cortex, in which indications of degeneration have also been found. This degeneration through the brain was traced in one case in which the disease of the anterior cornua was slight, the wasting was limited to the hand muscles, and the spastic paralysis began in the legs and then invaded the arms.* Such a case is almost a pure degeneration of the whole of the first segment of the motor path.

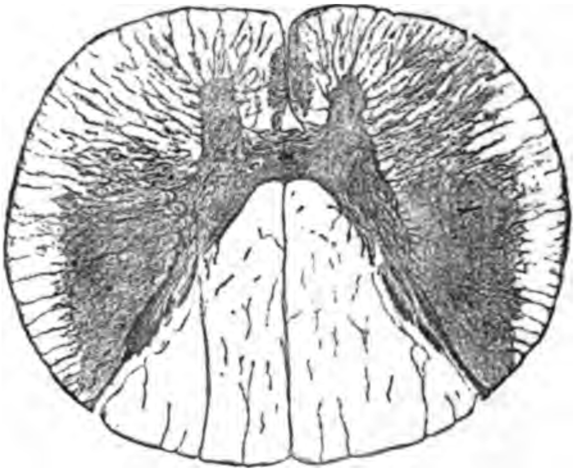

FIG. 128.—Sclerosis of the pyramidal tracts, lateral and anterior: dorsal region. From a case of muscular atrophy.

The degeneration of the pyramidal tracts is well shown in Fig. 128, from a case of progressive muscular atrophy. The anterior and lateral tracts are entirely degenerated, and the sclerosis stops abruptly at the outer margin of the lateral, but in front extends forwards, probably in the short fibres that connect the anterior cornu at different levels. In other cases, in which the anterior ganglion-cells were normal, the sclerosis has not been limited to the pyramidal tracts; the anterior columns have been also sclerosed in the lumbar region of the cord.[*] The direct cerebellar tract and posterior vesicular columns have been also found degenerated with the pyramidal tract, and an annular sclerosis of the periphery of the antero-lateral column has been found in several cases.[†] More frequent still is a combination with degeneration of the posterior columns, described more fully in the next section. In most of these combined cases the degeneration has lessened in the upper part of the cord. In one case, at least, the white columns of the cord have been found healthy. The probable significance of this fact has been already stated. It should be remarked, however, that there seems sometimes to be a tendency for the morbid process to extend beyond the pyramidal tracts in a manner that suggests a greater tendency for the secondary connective-tissue changes to take on an independent invasive activity than in other system diseases. (See also Ataxic Paraplegia.) Another point that deserves mention is the occasional occurrence of lateral sclerosis in association with insular sclerosis. In some cases of this character the degeneration of the pyramidal tracts is purely secondary, the result of the damage to the pyramidal fibres by an islet of sclerosis situated in some part of their course. But it does not appear that this explanation can be given of all cases. In some instances the sclerosis of the pyramidal tract appears to be independent and coincident. A similar association of insular and posterior sclerosis has also been observed; and such combinations receive additional emphasis from the fact that insular sclerosis may be accompanied by a primary atrophy of the optic nerve, precisely such as occurs in tabes. These facts make it probable that lateral sclerosis, although a system disease, is often one of less strict features than tabes, having some tendency towards chronic myelitis. We shall see that this is true also of ataxic paraplegia.

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* Kojewnikoff, 'Arch. de Neurologie,' 1883, No. 18.

The probable mechanism of the symptoms that give the dominant characters to the malady have been considered in the general account of the symptoms of disease of the spinal cord. The most important fact is that the degeneration, whatever its upward extent, always involves the lowest part of the pyramidal segment, because it seems to begin in this and ascend the fibres. Hence the intra-cornual termination must always be involved. This, it is assumed, is the structure that controls the muscle-reflex centre, and the consequent loss of control

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[*] As in a case recorded as one of spastic paraplegia by Hopkins ('Brain,' Oct., 1883), but this was an example of combined lateral and posterior sclerosis (ataxic paraplegia). An illustration of the changes in this case is given in the next section.

[†] Direct cerebellar and pyramidal tracts in a case by Minkowski, in which the disease rapidly succeeded syphilis; annular sclerosis by Westphal (see "Ataxic Paraplegia").

explains the excess of myotatic irritability and the progressive spasm, progressive by what may be termed a functional hypertrophy resulting from continuous over-action. When there is the rare consecutive wasting of muscles, we must conceive that the degeneration involves also the ganglion-cells and lower segment of the motor path; when there is coincident wasting of other muscles than those that are the seat of spasm, it would seem that some elements of this lower segment are the seat of a primary degeneration. The slighter muscular wasting without, or with only trifling, change in electrical irritability, is the expression of a slighter alteration in the nutrition of the cells and fibres, without actual destructive degeneration.

DIAGNOSIS.—The diagnosis rests on the combination of weakness, excess of myotatic irritability, and spasm,—on the gradual onset of these symptoms,—and also on the absence of indications of a focal lesion. An acute onset, occupying a few days or weeks, is *primâ facie* evidence of a lesion that takes the case out of the category of degenerative disease. In most acute cases there is other evidence of a focal lesion, extending, at some level, beyond the limits of the motor path. Such indications are initial impairment of sensation, or a girdle-pain. The latter indicates irritation of the posterior root-fibres at a certain level, and proves that at that level the disease extends beyond the limits of the pyramidal tracts. Spastic paraplegia is common after such lesions, but is secondary and not primary.

Although marked sensory symptoms, in a case that presents the symptoms and course of primary lateral sclerosis, are thus evidence that the sclerosis extends beyond the motor tracts, and that the case is not one of pure lateral sclerosis, it is doubtful what significance is to be attached to very slight sensory symptoms, such as slight subjective sensations of dull pain, formication, &c., when they exist alone. It is possible that they are due to functional disturbance in the sensory nerve-elements, and do not imply structural disease outside the motor area. They are not followed by any more pronounced sensory symptoms.

The absence of objective unsteadiness is the chief distinction from ataxic paraplegia (q. v.). When this is slight, it may be difficult to say in which class a case should be placed. There are no doubt intermediate cases in which there is a very slight degree of the additional lesion that exists in ataxic paraplegia, but the distinct objective character of the ataxy is the best criterion. Considerable muscular wasting in any part is commonly regarded as bringing the case into another category, that of "amyotrophic lateral sclerosis;" but some cases of the kind, in which the wasting is confined to a few muscles, resemble more closely the cases we are now considering than they do those with wide-spread and extreme muscular atrophy. These cases are further considered in the account of progressive muscular atrophy.

Primary spastic paralysis is not uncommon in those who are at the age and of the sex at which hysteria prevails, and there is no form of cord disease that is so often mistaken for hysterical paraplegia. The mistake is facilitated by the perfect muscular nutrition. But the mistake ought not to occur, as the peculiar extensor character of the spasm, lessening with flexion, is distinctive; nothing resembling it ever occurs in hysterical paraplegia. When the spasm is trifling or absent, as in slight and early cases, the diagnostic difficulty is much greater, and is increased by the fact that slight excess of myotatic irritability occurs in some cases of so-called hysterical paralysis. But this scarcely ever reaches the degree necessary to give rise to a true foot-clonus or a rectus-clonus. There may be a slight clonus produced through a voluntary depression of the foot of the patient in response to the passive flexion of the ankle, and readily recognised; or a true clonus may be obtainable during hysterical contracture, but apart from such contracture a true foot-clonus or a rectus-clonus deserves the greatest weight, as all but conclusive evidence of organic disease. I have known many mistakes in diagnosis, in which lateral sclerosis was mistaken for hysterical paraplegia owing to disregard of the evidence afforded by this symptom, but I have never known the opposite error from undue regard to this symptom. Moreover, an excess of myotatic irritability in so-called hysterical paralysis must depend on more than functional disease. There must be changes in nutrition, and consequent persistent defective control of the muscle-reflex centres. On the hypothesis that I have advanced, this control is exerted by the termination of the pyramidal fibres, *i. e.* of the upper motor segment, by the structures degeneration of which probably causes spastic paraplegia. A case is actually on record (the case of lateral sclerosis described by Charcot in 1865) in which an initial hysterical paraplegia, cured suddenly and relapsing on emotion, passed ultimately into lateral sclerosis.

When the arm and leg suffer on one side only, the disease may be mistaken for cerebral hemiplegia. There is not, however, any affection of the face, which, although theoretically conceivable, is, as a matter of fact, always absent. The limbs on the other side are never quite normal, but present slight symptoms—weakness and an excess of myotatic irritability—similar to those on the affected side, which indicate the nature of the malady.

The diagnosis of the congenital infantile form is only difficult when the observer is unaware of the occurrence of these cases. A slight degree of inco-ordination in the hands will usually be found if they are carefully watched while the patient takes hold of some object. The wide separation and irregular movement of the fingers is very characteristic. Chronic primary cord diseases are almost unknown in young children. Caries of the spine is, in them, the chief cause of paraplegia; and the definite onset of the paralysis, in a previously healthy child, is an absolute distinction from the cases of birth-palsy. Other diagnostic indications are described in vol. ii (Infantile Meningeal Hæmorrhage). A definite onset also distinguishes glioma of the pons, which may likewise cause spastic paralysis of arms and legs.

PROGNOSIS.—In primary lateral sclerosis there is some prospect of arrest, and even of improvement, if the disease has not reached an advanced stage. Actual recovery is rare, but does sometimes occur in early cases. When the spastic state is well developed and has lasted for some time, it is very rare for more than arrest to be obtained. This is perhaps due rather to the tendency of the disease than to its degree, since a similar condition that is secondary to a focal lesion often recovers perfectly; and many facts show that it is not likely that there is anything in the nature of the lesion to preclude the restoration of a normal condition, until dense connective tissue has replaced the nerve-elements. In the cases that improve most, it is possible that the disease is limited to the terminal structures. We have not, at present, any guide to the prognosis in an individual case, besides its duration and degree, except its observed tendency, especially under treatment. The prognosis of the infantile form is considered in vol. ii.

TREATMENT.—So far as drugs are concerned, the treatment is, to a large extent, the same as that of posterior sclerosis, already described. The drugs most useful are the same, but, unfortunately, their influence is less frequently appreciable. Nux vomica and strychnine have, however, to be given with caution, and in very minute doses, as they have a tendency to increase the spasm. In severe cases this is a most distressing symptom, and often not amenable to any influence. Bromide sometimes lessens it slightly, but even large doses of bromide have but a trifling effect. Indian hemp, belladonna, and calabar bean may be tried, but seldom have a distinct influence. One of them may be combined with arsenic, or whatever metallic agent is employed. Absolute rest is sometimes of service, and occasionally seems to produce improvement, which all treatment failed to effect while the patient was walking about. The avoidance of fatiguing exertion is a very important element in treatment, and often seems to permit medicines to do good, which before were counteracted. Rubbing is also beneficial in some cases. Its influence on the spasm is often very distinct during the process, and a long course of rubbing has produced a permanent improvement in the spastic condition. Upward rubbing seems to have more influence than kneading the muscles. If there is contracture of the calf-muscles, the foot should be pressed up while these muscles are rubbed. When there are facilities for it, the rubbing may advantageously be combined with sweating in the Turkish bath. In one case, of moderate degree, almost all the symptoms passed away after a long course of Turkish baths, arsenic being also given. The patient, who could at first walk scarcely half a mile, became able to walk several miles without fatigue, and the improvement was permanent. Electricity is useless in the pure disease. Faradism and all painful applications are harmful, stimulating further the already excessive excitability of the reflex centres. The constant current to the muscles, or from the spine to the muscles or to the feet in water, has no distinct influence in either lessening the spasm or improving the strength.

In the infantile form, drugs are useless. Rubbing is desirable, and can be efficiently performed by the nurse or mother. Carefully planned gymnastic exercises are also useful. The tendo Achillis is sometimes divided for the contracture of the calf-muscles, but the operation is useless and ought never to be performed. Supports help the child to walk somewhat sooner than it would without their aid, and so hasten improvement, but they should only be employed when there is sufficient power and control to make them useful by enabling the will to effect ordered movements.

ATAXIC PARAPLEGIA

(COMBINED LATERAL AND POSTERIOR SCLEROSIS).

The term ataxic paraplegia seems the most accurate clinical designation for a disease of the spinal cord which presents a combination of the symptoms of paraplegia and ataxy, and consists in combined disease of the posterior and lateral columns. Although its clinical features present some varieties, and may approach those of each of its constituent forms of disease, yet in the majority of cases the symptoms are uniform, and sufficiently characteristic to justify the distinction of the disease and its separate description. A few pathological observations have been published. The following account is based on these and on a series of cases that have come under my own observation, and of which the clinical characters were well marked.[*]

Causes.—Neurotic heredity is to be traced only in a small proportion of the cases—about one tenth. A history of syphilis is as rare as it is frequent in pure tabes. Males suffer much more frequently than females. The disease usually commences between thirty and forty, but I have known it to begin as early as nineteen and as late as fifty-two, while commencement at fifteen[†] and at sixty-one[‡] is on record. Exposure to cold is to be traced occasionally as an exciting cause. In one young lady the symptoms commenced after a season of balls, at which when heated she would habitually sit at open windows, and often sleep in clothes saturated with perspiration. In a few cases, severe exertion, such as excessive athletics, has been the apparent cause. A severe concussion of the spine has sometimes preceded the first symptoms by a few months. The disease may also follow great sexual excess. In many cases no cause can be traced, immediate or remote. Its general etiology thus resembles that of simple spastic paraplegia.

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[*] See Prevost, 'Arch. de Physiologie,' t. iv; Pierret, ib.; Babesieu, 'Virchow's Archiv,' Bd. lxxvi; Kahler and Pick, 'Arch. f. Psychiatrie,' Bd. viii; Westphal, ib., Bd. viii and x; Dana, 'New York Med. Record,' July 2, 1887; and Clarke, 'Brain,' 1890. The condition was described by Déjérine in 1884 under the name of "ataxo-paraplegic tabes;" see Ladame, 'Brain,' 1890, part lii, p. 530. I have not seen Déjérine's description, and did not know of it when the above chapter was written.

[†] Oppenheim, 'Neur. Cent.,' 1888, p. 647. Such early cases are generally isolated forms of "Hereditary Ataxy."

[‡] Suckling, 'Lancet,' 1886.

SYMPTOMS.—The onset of the disease is usually slow and gradual, two or three years passing before walking power is much impaired; it is rarely subacute, so as to reach a considerable degree in two or three months. The early symptoms resemble those of spastic paraplegia, with the addition of ataxic unsteadiness, which may be at first the most prominent symptom. The legs suffer first, often alone, or the arms may also be involved. The patient finds that he tires more readily than before, and also becomes unsteady on turning or walking in the dark, and these symptoms gradually increase. The weakness may be slight, but if all the movements are tested, a defect in power will be found in some, often most in the flexors of the knee and hip, and frequently more in one leg than the other. The patient is unsteady when he stands with feet together, and he tends to fall if the eyes are then closed. If the feet are bare, the irregular action of the muscles is shown by the movement of the tendons on the dorsum of the foot, as in tabes. In early cases, the inco-ordination is revealed by the patient's gait, which is distinctly unsteady. There is rarely the high movement and sudden descent of the feet often seen in tabes, but (as in many cases of tabes) the patient is unsteady, reels on turning, and has often to bring his foot suddenly to the ground to maintain his equilibrium. He may even have to steady himself with a stick, or to catch hold of some adjacent object, to save himself from falling. The ataxy is equally evident when he lies, and attempts, his eyes being closed, to touch some object with his foot.

The sensory and reflex symptoms present a marked contrast to those of tabes. Lightning pains are almost always absent; I have only met with them in one case, and in this they were a transient symptom. Sometimes there is a slight dull pain in the legs, felt especially on fatigue. Dull pain in the sacral region or in the spine is not uncommon, and is often an early symptom. The sacral pain, indeed, is sufficiently frequent to deserve special note. A girdle-pain is met with only in rare cases. As a rule there is no loss of sensation either on the legs or trunk. Hyperæsthesia is equally rare. Reflex action from the sole may be normal or increased; less commonly it is diminished. The cremasteric and abdominal reflexes are sometimes lost. The most striking difference from tabes, however, is in the condition of myotatic irritability, which, in the vast majority of cases, is greatly increased. The knee-jerk is quick and extensive; it can be obtained from above (see p. 16), and there is generally a distinct rectus-clonus. The foot-clonus is also commonly to be obtained. This myotatic excess persists and increases, and when the arms are involved they present symptoms similar to those in the legs—incoordination, weakness, and marked excess of the myotatic irritability.

There may be conspicuous ataxy of the hands, and a tendency to cramp-like spasm on an attempt to use them. The muscles in both arms and legs are usually well nourished.

Sexual power is often lost early in the disease. The sphincters may be impaired, sometimes early, but they also often escape. As in other diseases, inability to empty the bladder is apt to develop insidiously, without the patient's knowledge, and the organ may thus become habitually distended.

The iris usually acts to light, but I have seen loss of the light-reflex in two or three cases, and have once known accommodation to be lost, the action to light being normal. These symptoms occur chiefly in cases following syphilis. Optic nerve atrophy occurs only in rare cases, far less frequently than in tabes; I have seen one case in which there were indications of a retro-ocular axial neuritis. The external ocular muscles are unaffected as a rule, except for nystagmus, which often exists on movement of the eyes, but seldom when they are at rest.

Slight impairment of articulation is not uncommon; sometimes there are irregular tremulous movements of the face resembling those of general paralysis, and this in cases in which there is no mental change. Westphal has observed marked ataxy of the facial muscles. As a rule the mental state is either normal, or there is merely slight failure of memory.

As the disease increases, the muscular weakness and reflex spasm become more and more considerable; while the ataxy, after reaching a certain degree (not sufficient to prevent locomotion), necessarily sinks into the background as the paralysis increases. With the increased weakness, the aspect of the patient comes to be that of spastic paraplegia, described in the last section. Indications of unsteadiness may still be observed in isolated movements. The arms, if previously free, may begin to suffer, but sometimes they escape altogether. The motor weakness may go on to complete paralysis. This is quickly reached in some cases, but so slow is the usual progress of the disease that one patient, in whom the disease was at no time absolutely stationary, was still able to stand, after eight years. In spite of the progress of the weakness, sensation remains unimpaired, and the cranial nerves do not suffer.

The symptoms present, in some cases, variations from this type. Sensation on the legs may be impaired, and the knee-jerk may be lost. These cases are, however, very rare, and are probably cases of true tabes with lateral sclerosis added. Other cases occur in which a girdle-pain, impaired sensation, &c., suggest extensive changes in the cord, although the state of the legs is that common in the disease. Such cases, as we shall see, are probably examples of chronic myelitis, or are intermediate between that and the disease now under consideration.

Ataxic paraplegia has little tendency to cause death. Indeed, the fatal cases have, for the most part, been untypical, and do not convey an accurate idea of the characters of the disease. The chief danger to life is from the accidents common to all chronic spinal affections, —bedsores, and especially kidney disease from undiscovered or untreated imperfect action of the bladder.

Among complications the most important are mental changes resembling those of general paralysis of the insane, of which indeed this combined sclerosis may form part. Slight muscular atrophy sometimes occurs. Arthritis of doubtful significance has been once observed. A patient of Westphal's presented derangement of the sympathetic, and died from peculiar spasm of the muscles of respiration. Visceral crises, however, are practically unknown.

PATHOLOGICAL ANATOMY.—In all cases the spinal cord has presented sclerosis of both posterior and lateral columns; but the precise extent and degree of the degeneration are subject to considerable variations. As a general rule, the sclerosis of the posterior columns differs from that of tabes in two particulars. First, it is not more intense, and often it is less intense, in the lumbar than in the dorsal region of the cord. Sometimes, indeed, as in the case shown in Fig. 129, in the middle and lower parts of the lumbar region, the posterior columns may be free from sclerosis, although it is considerable in the dorsal region and at the junction of this with the lumbar enlargement (B). The second difference is that the sclerosis has not that special intensity in the root-zone of the postero-external column which characterises the lesion of tabes. In rare cases the whole posterior column in the lumbar region is diseased. The part of the external column near the commissure and near the neck of the posterior horn usually remains free. Sometimes the degeneration does not extend up to the posterior surface of the cord; for instance, as in the case figured, it affects chiefly the middle three fifths of the posterior columns. When the degeneration is considerable, the posterior median columns, in the upper part of the cord, may present the usual ascending degeneration of secondary origin. When the lesion is slight in degree in the lower half of the cord, there may be only a diffuse degeneration of the columns in the cervical region, similar to that below, and not the more intense limited affection of the median part which occurs when there is a typical ascending degeneration from a considerable lesion of the column lower down.

The degeneration in the lateral columns is also variable in extent and position, and is often not strictly "systemic" in character, *i.e.* is not strictly limited to a single system of fibres, although the pyramidal tracts are chiefly affected. In one or two cases, indeed, the whole pyramidal tract, and this alone, has been degenerated; but more often the sclerosis, while intense in this tract, also extends in front of it into the mixed zone of the lateral columns (Fig. 129, A, B). A similar extension, however, is usually met with in the sclerosis that attends degeneration of the anterior cornua; compare Fig. 127. The lateral limiting layer, between the pyramidal tract and the grey matter, may be also invaded. The direct cerebellar tract often escapes, as in Fig. 129, A (B is below its level of origin), but it is affected in some cases. Occasionally a zone of sclerosis has existed in the whole periphery of the cord, extending deeply into the lateral column in the position of the pyramidal tract. This tract has been found diseased, in some degree, in all cases, and in several the extent of its degeneration has been greater in the lower part of the cord than in the upper. The direct (anterior) tract may be affected as well as the lateral, and is commonly involved when the lesion is considerable in the upper part of the cord. As in most other degenerative diseases of the cord, there may be some increase of connective tissue in the unaffected columns. As a rule no morbid change has

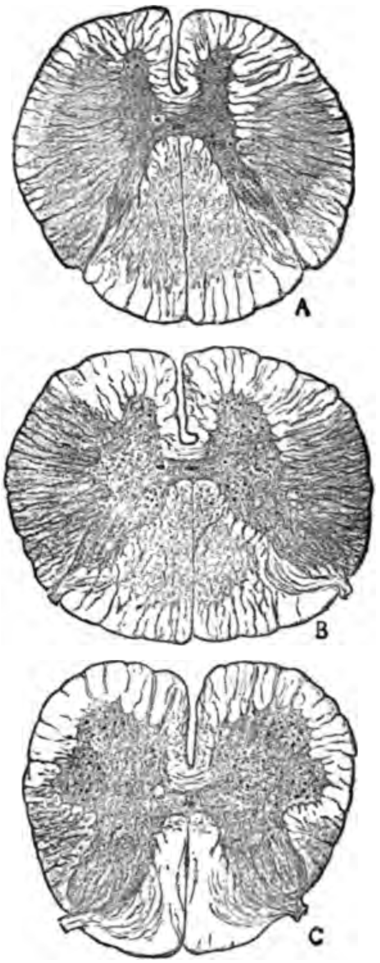

FIG. 129.—Ataxic paraplegia, combined lateral and posterior sclerosis. A, upper dorsal; B, last dorsal; C, mid-lumbar. The posterior columns are free from sclerosis in C, except in their anterior parts; in B the disease involves the middle and anterior parts of both post.-med. and post.-ext. columns except in the neighbourhood of the neck of the horn; in A the sclerosis is slighter and is confined to the middle three fifths of these columns. The sclerosis of the lateral columns in C is limited, on the right, to the pyramidal tract, on the left it extends in front of this; in B it is very dense in the whole lateral column, involving not only the pyramidal tract but the limiting layer, and part of the "mixed zone;" in A it is similar in extent but slighter in degree. There is some increase of tissue throughout the anterior columns, and a focus of sclerosis near the anterior fissure, on the right in A and B, on both sides in C.*

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* I am indebted to Mr. J. Hopkins for the opportunity of drawing these sections. The patient was a man aged twenty-one, in whom weakness of the legs commenced at twenty, after a wetting, improved, and then slowly increased. He was admitted

been recognised in the grey matter or in the membranes. In one case only was there slight meningitis. Disease of the ascending root of the fifth has been found in some cases;[*] in one there was also atrophy of the cells of the oculo-motor nucleus, and the case thus differed considerably from the type. The muscles have been found normal.

PATHOLOGY.—The double lesion in the posterior and lateral columns supplies an explanation of the two sets of symptoms which characterise the disease. The inco-ordination must be referred to the disease of the posterior columns, which, greatest in the dorsal region, involves there the fibres that conduct impulses from the muscles, probably to the cerebellum.[†] The effect must be to lessen the cerebellar guidance, and this explains the resemblance of the inco-ordination to that in cerebellar disease. The fact that the disease does not extend into the lumbar root-zone accounts for the integrity of the muscle-reflex action, and enables us to understand this marked difference from tabes. Disease of the direct cerebellar tract probably has an influence similar to that of the posterior median columns (see p. 215). That interruption of the conducting path in the cord will produce inco-ordination is, as we have seen (p. 205), well established. The sclerosis in ataxic paraplegia occupies also the region of the cord in which the short vertical fibres run, connecting the posterior grey matter at different levels, and it is possible that the damage to these fibres may contribute to the ataxy.

The only recognisable lesion that can be regarded as the cause of the paralysis is the degeneration of the pyramidal tract. The statements made regarding spastic paraplegia apply also to the disease, and need not be repeated. The lesion has been found greater on the side on which one leg was weaker than the other. The variations in the amount of disease, which does not always correspond to the degree of palsy, are probably due to the fact that the latter may be caused by degeneration of the termination of the fibres, which may be much greater than that of the fibres higher up the cord. In most cases that have been examined, the visible disease was greater in the lower than

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a year after the onset, with considerable weakness of the legs, a reeling, unsteady gait, increased knee-jerk, foot-clonus, but with no anæsthesia, wasting of muscles, pains, or affection of the arms. The symptoms slowly increased, paraplegia became absolute, and the spasm very intense, sometimes flexor and sometimes extensor. There was a doubtful impairment of sensibility to touch on the legs, but no loss to pain. The sphincters became affected, bedsores formed, and the patient died two years after the onset. Fuller details will be found in 'Brain,' October, 1883, p. 383.

[*] Oppenheim, 'Neur. Cent.,' 1888, p. 647.

[†] Much evidence has been obtained of the connection of the posterior median columns with the cerebellum through the grey matter of its bulbar nucleus, and also of the effect of interruption of these columns in the dorsal cord, in causing inco-ordination like that of cerebellar disease. Besides the experiments of Bechterew on the latter point already mentioned (p. 215), a recent important investigation into the connections of the cerebellum fully confirms that with the post-pyramidal nucleus (Brosset, 'Contrib. à l'étude des Connexions du Cervelet,' Paris, 1891).

in the upper part of the cord, increasing from above downwards, a fact which suggests that the maximum lesion is at the lowest part—in the terminal ramification within the grey matter.

The chief pathological difficulty arises from the fact, which we have already considered in connection with spastic paraplegia, that the morbid process often extends beyond the limit of the tracts chiefly diseased. It tends to assume a diffuse character, so that it has been regarded by some as rather a chronic myelitis than a system degeneration. It may occupy an intermediate position, and illustrate the fact that there is a close connection between the primary decay of nerve-elements and the growth of the connective-tissue elements that replace the former. In a pure secondary degeneration this growth is limited to the tract affected, but even in tabes it may manifest some degree of independence, which seems to be still greater in ataxic paraplegia. Sometimes, indeed, the primary morbid tendency may be double, involving both the nerve-elements and interstitial tissue. If it so far preponderates in the latter as to cause considerable changes outside the tracts specially diseased, and marked symptoms, such as a girdle-pain or loss of sensation, the condition is really one of chronic myelitis, and the cases should be so classified. This is probably the true solution of the problem, which has been much discussed, whether the malady is or is not a system disease. It is so in typical cases, but it is on the border of the group of system diseases, and intermediate cases connect it with diseases of diffuse character and non-systemic nature.

DIAGNOSIS.—The diseases from which ataxic paraplegia has to be distinguished differ according to the stage of the disease at which the diagnostic problem presents itself. The dominant symptom in the early period is the inco-ordination; in the later period, the spastic palsy. Hence the disease in the early stage is liable to be confounded with pure locomotor ataxy, but the condition of the knee-jerk, lost in the one, excessive in the other, is distinctive. In the rare cases of early tabes in which it is not lost, it is never increased. In tabes, there is not the loss of power which will always be found in ataxic paraplegia, and when a clonus and extensor spasm are added, there should be no risk of confusion; but it is necessary to emphasize the distinction, because cases with much unsteadiness are frequently described as tabes with preserved knee-jerk. From primary spastic paraplegia the diagnosis depends on the presence of inco-ordination, or in a history of it, if voluntary power has become too slight to be susceptible of derangement. Ataxic paraplegia is spastic paraplegia *plus* inco-ordination. If the ataxy is considerable, as it often is, the distinction is easy enough, as experience shows that it is chiefly with tabes that such cases are confounded. If slight, it may be difficult to say in which class the case should be placed; the best criterion is that mentioned in the account of spastic paraplegia, the distinctly objective character of the unsteadiness. The so-called "hereditary ataxy" presents a close resemblance to ataxic paraplegia, and is, indeed, as we shall see, intermediate between this disease and true tabes. It is distinguished by its occurrence in several members of the same family, and by the common loss of the knee-jerk; while the presence of nystagmus, of some impairment of articulation, and of other symptoms described in the account of the disease, distinguish the isolated cases that are sometimes met with.

The distinction from diffuse or focal chronic myelitis depends chiefly on the extent of the symptoms in the former, and their limitation in the latter. The subacute forms of myelitis are also distinguished by their more rapid onset.* The greatest difficulty is presented by cases which have a subacute onset, and the distinction from a local myelitis involving the posterior and lateral columns may be very difficult. It depends chiefly on the progressive tendency of the symptoms, which contrasts with the regressive tendency of myelitis. It is possible that when there is a degenerative tendency, a focal myelitis may set up a progressive degeneration in the columns concerned, as it certainly may set up a degeneration limited to the posterior columns.

A tumour in the middle lobe of the cerebellum may cause unsteadiness, closely resembling that of some cases of ataxic paraplegia, and it may also cause weakness of the legs with increased knee-jerk, from the pressure on the pyramidal fibres as they pass through the pons. In such cases, indeed, we probably have the two elements of the disease, produced in a different manner, by lesions of different position. We have a degeneration of the lateral columns which is secondary instead of primary, and we have disease of the co-ordinating centre instead of interruption of the path to it. But the weakness in the legs is never great in cerebellar tumour, and special symptoms of this are never absent. Occipital headache, vomiting, and optic neuritis are present in most cases. In a case in which the pons is compressed, some cranial nerves are usually also damaged.

PROGNOSIS.—The prognosis in ataxic paraplegia is similar to that in the spastic form, and depends on similar considerations, which need not be here repeated.

TREATMENT.—The treatment of ataxic paraplegia is the same as that of the allied diseases, and especially of spastic paraplegia, to the account of which the reader is referred.

PELLAGRA.

Pellagra is an endemic malady which, though fortunately unknown in this country, deserves mention here on account of the incidence of its effects on the spinal cord. Its chief anatomical lesion seems to be a degeneration of the lateral and posterior columns of the cord, corresponding closely to that of ataxic paraplegia, and, as in that disease, with a greater affection of the lateral than of the posterior columns. It differs, however, in the fact that some atrophy of the large nerve-cells of the anterior cornua is common, and also in the constancy with which chronic inflammation of the pia mater is met with, sometimes accompanied by the formation of bony plates in the arachnoid.

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* For some instructive notes on this point in diagnosis see Dreschfeld, 'Brain,' January, 1888.

The *Cause* of the disease appears to be the action, on the elements of the spinal cord, of an organised virus (or of some product of this) which is taken into the system with diseased maize. The virus has been thought by some (as Lombroso) to be a fungus growing in the maize; by others to be specific micro-organisms of other than fungoid nature, present in the maize, which develop in the system and produce there the toxic agent that has a special action on the nerve-elements (Belmondo).

The *Symptoms* resemble very closely those of ataxic paraplegia. There is weakness in the legs, with increased myotatic irritability going on to spasm; unsteadiness of movement, especially in the early stages, sometimes accompanied by tremor. Sensibility may be normal, or the perception of touch or pain may be increased or lessened. Belmondo, from an important series of investigations,* has arrived at the conclusion that the symptoms depend on a primary systemic degeneration of the nerve-elements of the pyramidal tracts and posterior columns, determined by the influence of the toxic agent. The course of the disease is chronic, but sometimes an intensely acute stage comes on, with symptoms of spinal meningitis,—a condition to which the term " typhus pellagrosus " has been applied. After death in this state there are often found commencing acute myelitis, in addition to the inflammation of the membranes, and also swelling of the intestinal lymphatic glands, with signs of enteritis,—indications of an acute blood-state which justifies the view that the affection is, primarily, a toxæmic malady.

"HEREDITARY ATAXY"

(FRIEDREICH'S DISEASE, HEREDITARY ATAXIC PARAPLEGIA).

The so-called hereditary ataxy is a form of ataxy, or rather of ataxic paraplegia, depending on combined posterior and lateral sclerosis, which occurs in families, and differs further from the common forms of tabes and of ataxic paraplegia in the early age at which it always commences, and in the presence of certain additional symptoms. It is often termed "Friedreich's disease," because this physician first described the characteristic features of the malady and the lesion in the posterior columns.[*]

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* 'Rev. Speriment.,' 1889-90, xv and xvi.

The dependence of the disease on a congenital tendency is clearly shown by its usual occurrence in families. But direct inheritance has been traced in only a few instances. In one, the mother of the family affected, and *her* mother, both suffered from the disease. That a general neuropathic inheritance is also sometimes effective is shown by cases in which there is a history of other neurotic diseases, such as insanity or epilepsy, in collaterals or ancestors. Two brothers who were affected had another brother epileptic, and a sister suffered from repeated attacks of chorea. Alcoholism in parents has been supposed to be influential, but the evidence of this is not strong. Consanguinity of parents has existed in some instances, and has doubtless intensified the morbid tendency in these cases.

The family tendency of the disease is shown by the affection, in most instances, of brothers and sisters. Thus sixty-five cases were distributed in nineteen families, giving an average of rather more than three to each. The number affected in one generation has varied from two to eight. In the case in which the mother and grandmother suffered, an uncle and seven children were also affected, making ten in one family. Few isolated cases have been recorded, but they certainly occur. Probably the nature of the affection has not been suspected on account of the isolation. In many of these cases (to judge from my own observations) this feature extends beyond the disease; the patient is an only child, or the only one of the sex in the family. Thus one patient was the only daughter, and her four brothers were healthy.

The two sexes present nearly equal liability; males slightly preponderate (thirty-five males to thirty females). In some families the two sexes have suffered equally, but in others the disease has shown a marked tendency to affect one sex. Thus in one family of nineteen, the two males suffered, and the seventeen females escaped. In another instance, recorded by Musso, a brother and sister (the offspring of a melancholic mother) married healthy individuals; the brother had three daughters affected out of seven living children; and of the sister's children three sons were diseased. A curious fact is that three of the brother's and four of the sister's children were born dead.

The age at which the first symptoms are recognised has varied between two and twenty-four years.[†] The seventh and eighth years of life are those in which disease most often begins; and next comes the period of puberty, from twelve to sixteen. In isolated instances the onset is generally later than in the grouped cases. It begins somewhat earlier in males than in females, and often commences about the same period in the same family.

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[*] Friedreich's first account was given to a medical society in 1861, and published in 'Virchow's Archiv' (Bd. xxvi and xxvii) in 1863. Further cases were published by him in 1876 (ib., Bd. lxviii and lxx), and a collection of 57 cases is given by Dr. Everett Smith, 'Boston Med. and Surg. Journal,' Oct. 15, 1885. See also Ormerod, 'Brain,' Jan., 1888; Déjérine, 'Méd. Moderne,' 1890, No. 25, and 'La Sem. Méd.,' 1890, Nos. 11, 12; and especially Soca, 'Thèse de Paris,' 1888 (who has collected 165 cases—many, however, doubtful), and Ladame, 'Brain,' 1890, pt. lii, where a full bibliography will be found.

[†] In the family recorded by Everett Smith, the father presented symptoms of ataxic paraplegia at the age of sixty-six, coming on gradually after an attack of

Immediate causes can rarely be traced; preceding acute diseases may have facilitated the onset, but can scarcely have done more.

SYMPTOMS.—The first and chief manifestation of the disease is a gradual impairment of co-ordination, first in the legs and afterwards in the arms. Initial pains scarcely ever occur. The ataxy is shown by unsteadiness in standing and walking, at first slight, but slowly increasing, until the feet have to be placed wide apart in standing, and the patient reels in walking like one under the influence of alcohol. The feet are not often raised too high, unless when an unusually long step is taken. Closure of the eyes causes a considerable increase in the unsteadiness in some cases; in others it has but little influence. Children often first show the affection by the readiness with which they stumble and fall. The impairment of movement in the arms is of a similar character, but usually commences some time after that of the legs. There is irregularity in the voluntary movement of the arms and fingers, and the ataxy has often a distinctly jerky character. In most cases the power of the muscles is at first unimpaired; their nutrition is good, but the myotatic irritability is lost. In most cases the knee-jerk has been found absent as soon as the patient came under observation; in one case it disappeared after the other symptoms had set in; in another it was normal on one side, absent on the other. In several there has been no change long after all the other characteristic symptoms had manifested themselves; very rarely it has been increased, chiefly in untypical cases.

As the disease progresses, some jerky irregularity develops in the movements of the neck and head, so that the head presents slight unsteady movements, sometimes like an irregular tremor, when it is supported by the muscles. Speech is also impaired; syllables are elided, and there may be, with this, an occasional hesitation. There is no twitching of lips, but occasional jerky movements have been noted in the tongue. The affection of speech is not often an early symptom. It may not be noticed until three, five, or ten years after the onset of the other symptoms. In most cases (but not in all) there is nystagmus when the eyes are moved laterally or upwards. Sometimes the movement is slower than in most forms of nystagmus, and it is rarely present when the eyes are at rest, directed straight forwards. This symptom may come on after those in the limbs, but careful examination will often reveal it in the early stage. Paralysis

rheumatism produced by exposure to cold. In the age of the sufferer this case stands alone in the history of the disease, and is not included in the figures given. The case may be a mere coincidence, or may show that a latent predisposition may persist through life.

of the ocular muscles is very rare; in one case there was strabismus with double vision,[*] and slight diplopia sometimes exists for a time and passes away. Optic nerve atrophy never occurs. The pupils are usually normal, but several cases have been recorded in which there has been loss of the light reflex. Whenever this condition exists, evidence of hereditary syphilis should be most carefully sought for, as it is probable that many of the cases in which this loss was met with were cases of true tabes, with some involvement of the lateral columns, occurring in young persons, and should not be classed with the disease in question.

Sensory symptoms are very variable. Lightning pains and any severe pains are extremely rare, though in one or two recorded instances they have been severe; but slight dull or rheumatoid pains in the legs are not uncommon. Sensibility has been quite normal in many cases even of severe degree; in others there has been slight anæsthesia in the legs, early in some instances, late in others. In one case there was delay of conduction, such as is met with in tabes;[†] rarely sensibility to pain and temperature is impaired. The sense of posture of the limbs has been found normal in several cases in which it has been examined. The electro-sensibility of the muscles is said to be sometimes lessened. Increased sensitiveness to pain is occasionally met with. Reflex action from the sole is usually preserved, but may be lost when there is anæsthesia. The cremaster reflex is often lost. On account of the age of the patients little is known of the condition of sexual power, but it is certainly often absent. Menstruation usually becomes irregular and ceases. The sphincters, as a rule, are unaffected. There is no tendency to trophic changes in the skin or joints.

Although muscular power is commonly normal at first, and may be normal even when the ataxy is considerable, it usually becomes impaired as the disease progresses, and sometimes weakness comes on, with the ataxy, at the onset of the disease. The loss of power is always greatest in the legs, and may be confined to them. The flexors suffer more than the extensors, and the weakness in the flexors of the ankle may permit some degree of talipes to occur. The loss of power may be ultimately great, although it rarely amounts to absolute paralysis. Slight wasting of the muscles may occur in the later stages of the disease, attended with only trifling depression of the electrical contractility; in two recorded cases, however, a brother and sister, there was great atrophy of muscles with altered electrical reactions.[†] Lateral curvature of the spine, and talipes equinus or equino-varus of the feet, are common as later results of the muscular weakness, developing under the influence of posture; and contraction of the flexors of the knees has also resulted.

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[*] Charcot, 'Prog. Méd.,' 1887, No. 23.

[†] Déjérine, 'Méd. mod.,' 1890, No. 25.

Visceral crises do not occur. Frequency of pulse has been noted, however, in many cases, and curious vaso-motor symptoms (flushing, œdema, sweating, polyuria, and salivation) were present in one of Friedreich's cases. There is no mental change that can be regarded as part of the disease, although imbecility has co-existed (Power).*

The isolated cases may correspond closely to the type, presenting the same nystagmus, weakness, and unsteadiness; many of these cases, however, differ somewhat, although sufficiently characteristic in their main features to justify their inclusion. Speech more often escapes. I have once met with a congenital lisp. In some the symptoms are slight, and have developed late. A difficulty in micturition may precede other symptoms. There are also cases, sporadic and grouped, in which coarse tremor is conspicuous, and the condition might be regarded as one of simple tremor, were it not for the presence or development of other symptoms. For instance, one man had clipping speech "all his life;" between forty and fifty, difficulty in writing came on, owing to a peculiar spasmodic tremor in both arms, brought on also in the legs by exertion; at fifty, unsteadiness on walking was added. There was a history of a similar affection in several relatives.

The rate of progress of the malady varies much, even in different members of the same family. One, in whom the disease commenced last, may be unable to walk while another who suffered sooner may be still in the early stage of the disease. Now and then the disease is stationary for many years. The duration of the malady is correspondingly variable. It is always long, even more than thirty years, and in many instances it has not apparently shortened life. On the other hand, death may occur at the end of ten or twelve years. The end has generally come from intercurrent affections, rarely from exhaustion. Acute myelitis has also caused death (Everett Smith).

PATHOLOGICAL ANATOMY.—The lesion in hereditary ataxy is that of ataxic paraplegia and of tabes combined. There is degeneration in the lateral column, often also in the anterior column, such as occurs in ataxic paraplegia; but the sclerosis of the posterior column is more intense than in that disease, and it is also more extensive, especially in the lumbar region. It is similar to the degeneration of tabes, and agrees with this also in that the posterior nerve-roots are usually affected, whereas in ataxic paraplegia they almost invariably escape.

The distribution of the lesions shows a close correspondence in different cases, as will be seen by a comparison of the figures here given, which are from three different cases of the disease. The sclerosis of the posterior columns may be complete throughout the cord, with the exception of a narrow band near the neck of the cornu, which always remains but little damaged. In the lumbar region it is usually intense up to the head of the horn and the posterior surface of the cord; occasionally, however, it is somewhat less intense in this region than it is higher up, extending, nevertheless, into the root-zone. In the cervical region the sclerosis may also be general, or it may be greatest in the root-zone and in the posterior median columns. The cervical root-zone never escapes, as it often does in tabes.

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* An excellent analysis of the symptoms, &c., is given by Crozier Griffith ('Trans. Coll. Phys. Philadelphia,' 1888); and by Ladame ('Brain,' 1890, pt. 52).

The degeneration of the lateral columns always involves the pyramidal tracts, and is most intense in their position. It is not, however, limited to them. It usually extends outwards to the periphery of the cord, even where the pyramidal tract does not reach the surface, thus involving the direct cerebellar tract; and it also extends forwards at the surface, so as to constitute a band of annular sclerosis, which varies in thickness in different parts of the cord and in different cases (Figs. 130, 132). In the inner part of the anterior column there may be a distinct

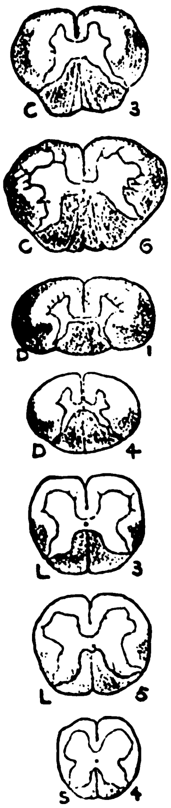

FIG. 130.—Hereditary ataxy; distribution of degeneration in the white columns, indicated by the dotted shading. (After Friedreich.)

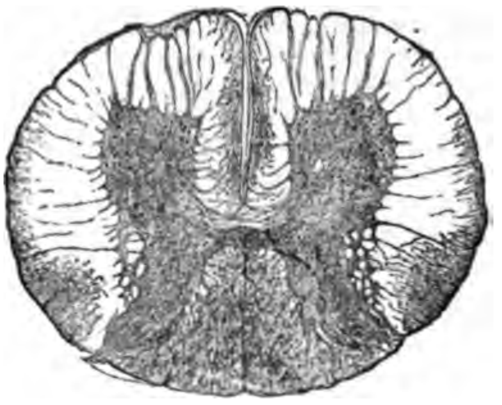

FIG. 131.—Hereditary ataxy; section of spinal cord at first lumbar segment. Sclerosis of the whole of the posterior columns except a narrow zone adjacent to the neck of the horn. Degeneration also of the lateral pyramidal tracts, and, in front of this, slight degeneration in the superficial layer of the lateral column. Adjacent to the anterior median fissure there is also a zone of sclerosis of the anterior pyramidal tract, which extends, in this cord, into the lumbar region.*

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* I am indebted for this section to Dr. Everett Smith, who has published the case in the 'Boston Med. and Surg. Journal,' Oct. 15, 1885. The patient was one of three sisters affected with the disease, the brothers being healthy. The father suffered from ataxic paraplegia late in life, as mentioned on p. 462. In the case from which the drawing is made, the inco-ordination began in the legs at the age of nineteen, and in the arms soon afterwards, and was quickly followed by loss of power, which gradually increased to almost universal paralysis, with muscular contractures (talipes equino-varus, &c.) and considerable loss of sensation. There was nystagmus, affection of articulation, and some mental dulness. Death occurred at the age of forty.

area of degeneration in the position of the anterior pyramidal tract. This is seen in Fig. 131, in which this tract extends unusually low in the cord, and is distinctly degenerated in the upper part of the lumbar enlargement. Atrophy of the posterior vesicular column has been found associated with that of the direct cerebellar tract. Slight abnormal appearances have been seen in the nerve-cells of the anterior cornua in some cases, but, as a rule, these are normal, and the grey matter presents no other disease. It is very likely, however, that future observations will reveal the occurrence of changes in the nerve-cells of the posterior horns, such as are met with in tabes.

The pia mater over the posterior columns has been found thickened. The posterior nerve-roots usually present some degeneration of their fibres; this may be partial (Fig. 133, B), or may amount to total destruction of the fibres (as at C). The peripheral nerves in the limbs have been found normal.[*]

Some general shrinking

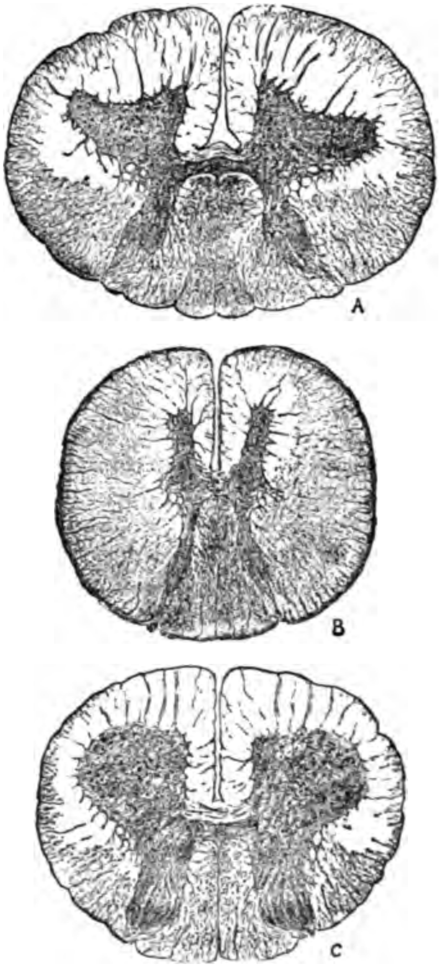

FIG. 132.—Hereditary ataxy. A, cervical; B, dorsal; C, lumbar regions of the cord. The posterior columns are sclerosed in their whole extent, except in the vicinity of the neck of the horn in the cervical and lumbar regions, the degeneration being rather less intense in the lumbar than in the other parts. In the antero-lateral columns there is an annular zone of sclerosis in the periphery of the cord, widening in the region of the pyramidal tract. In front of this tract the zone is widest in the dorsal region, and trifling in degree in the lumbar. The grey matter is but little affected.[†]

[*] Friedreich, 'Virchow's Archiv,' Bd. lxx, p. 145.

[†] For the sections from which the drawing is made I am indebted to Dr. Pitt (who prepared them) and the late Dr. Moxon, under whose care the patient died in Guy's Hospital. The case (full details of which will be found in a recent volume of the 'Guy's Hospital Reports') is one of a series of five cases (four brothers and a sister) which I reported some years ago to the Clinical Society ('Clin. Soc. Trans.,' vol. xiv, 1881, p. 1).

and induration of the pons and medulla were found by Friedreich, with atrophy of the cells of the post-pyramidal nucleus and some degeneration of the restiform bodies, but none of the anterior pyramids. Corpora amylacea were present in the hypoglossal nerves.

Atrophy of the cerebellum was found in one case; the cells of Purkinje were diminished in numbers, but showed no sign of atrophy or degeneration.*

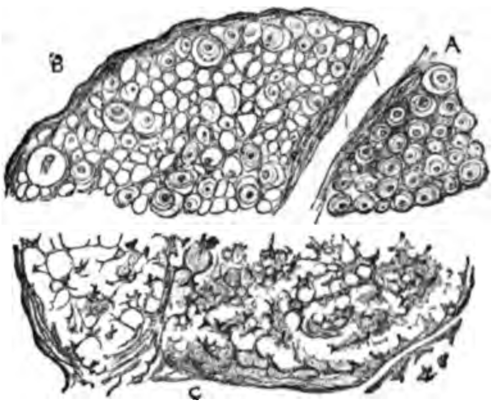

FIG. 133.—Hereditary ataxy; degeneration of posterior nerve-roots (from the same case as Fig. 124). A, normal anterior root, for comparison. B, posterior root, partially degenerated; a few normal nerve-fibres are seen, but most of the spaces which should contain nerve-fibres are empty. C, posterior root, totally degenerated, probably by a more acute process than that of B, since there is some destruction of the septa and increase of amorphous connective tissue. The products of degeneration are rendered invisible by the mode of preparation. (Compare C with the similar changes in a totally degenerated peripheral nerve shown in Fig. 54, B, p. 132.)

PATHOLOGY.—The disease apparently occupies a clinical and pathological position between the combined sclerosis described as ataxic paraplegia and simple tabes, resembling the former in the common affection of the lateral and posterior columns, and the consequent weakness with ataxy—resembling it also in the imperfect limitation of the changes, and in the intensity of those in the middle of the posterior columns in the dorsal region; but it differs from this, and resembles tabes, in the degree of affection of the posterior columns in the lumbar regions, the extension in slight degree into the root-zones, and the affection of the posterior roots; with these the loss of the knee-jerk and the affection of sensation are apparently connected. The isolated cases, occasionally met with, present the same combination of spinal symptoms. The precise origin of the affection of articulation and of the nystagmus has not yet been traced.

The age at which the disease commences, long before the period at which ordinary degenerations occur, and its family grouping, suggest that its ultimate cause is a congenital tendency of development, by which the affected elements have a briefer period of vital endurance than the other tissues of the organism. Möbius has suggested that there is an actual arrest of development of these structures, but it is evident that their development suffices for perfect function during the early part of life, and that their functional capacity undergoes subsequent failure, which can only be due to a process of structural change. It is possible that we have here, again, a double morbid process, and that a tendency to early degeneration in the nerve-elements is associated with a converse tendency to overgrowth of the interstitial tissue, analogous to that which we have in the muscles in Pseudo-hypertrophic Paralysis (*q. v.*). In this connection it is important to note that, in one case, the neuroglial overgrowth suggested a pure increase of the normal tissue, rather than a result of inflammation; and the peripheral sensory nerves, although not degenerated, contained a considerable number of embryonal nerve-fibres in the fasciculi.* Déjérine would regard the neuroglial increase as the sole element in the morbid process; but the fact last mentioned, as well as the preponderant affection of certain tracts, makes it improbable that this exclusive view is correct.

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* P. Menzel, 'Arch. f. Psych.,' Bd. xxii, H. 1, p. 160.

DIAGNOSIS.—In most cases the inco-ordination is sufficiently predominant to suggest that the disease is a form of ataxy, and the unsteadiness of the head, the affection of articulation, the nystagmus, and the age at onset, suffice for the distinction from ordinary tabes. The common form of ataxic paraplegia bears a close resemblance to this disease, a resemblance that is more than superficial, although the excessive knee-jerk and foot-clonus, almost always present in ataxic paraplegia, are usually absent in hereditary ataxy; much spasm is always absent, and nystagmus is common. The difference in the state of myotatic irritability is, as we have seen, not absolute; cases of combined lateral and posterior sclerosis occur, of tabetic type, in which the knee-jerk is lost, and in at least one case of hereditary ataxy the knee-jerk has been excessive. The age of onset and family multiplicity may decide the question, but the isolated cases, which are by no means rare, commencing soon after puberty, are not distinguishable from the hereditary disease; they seem to be analogous to the isolated cases of pseudo-hypertrophic paralysis. A greater difficulty is presented by cases of slight tabes in children, the subjects of inherited syphilis, in whom it is not uncommon for some weakness of the legs to co-exist, or speech to be disordered, in consequence of some early lesion of the brain. The loss of the iris-reflex should suggest inherited syphilis, and other indications of this will often decide the nature of the case.

Disseminated sclerosis presents inco-ordination, nystagmus, and impaired articulation, but the ataxy of the arms differs in the wide range and violent character of the disordering jerks which characterise this disease; while simple unsteadiness of the legs is very rare, and the course of the malady is more rapid. The affection of speech differs from that of hereditary ataxy in being simply " staccato," with undue separation of syllables, which are rarely run together. The cases are always isolated.

Cerebellar tumour and hereditary ataxy can hardly be confounded, in spite of the fact that the unsteadiness in walking is very similar in the two diseases; the common affection of the arms in the one, and the conspicuous head symptoms (severe pain, optic neuritis, &c.) of the other, sufficiently distinguish them. It is very common for the tremor of the head to be at first ascribed to mere "nervousness," and also for the isolated form, in girls, to be regarded as hysterical. The presence of nystagmus should at once decide the question; it is absolute proof of definite disease.

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* Auscher, 'La Semaine Méd.,' 1890, No. 32.

PROGNOSIS.—The prognosis in every case is very serious, since the disease, being a developmental affection, is essentially progressive; but life may be prolonged for many years, and in slight cases the malady may interfere comparatively little with the patient's occupation. Thus I have known a man, with very marked symptoms, follow his business as a tradesman for many years. The only guide to individual prognosis is the observed rate of progress, which has little relation to the age at which the symptoms commence.

TREATMENT.—As in other diseases that depend on a congenital tendency, treatment is almost powerless. The measures recommended for ordinary locomotor ataxy (apart from those suggested by the special symptoms of tabes) are those most suitable to the hereditary form. Arsenic, phosphorus, and nitrate of silver deserve a trial; now and then they seem to check the progress of the disease for a longer or shorter time, but its individual tendency determines its course, almost irrespective of treatment.

SIMPLE SENILE PARAPLEGIA.

This name seems the most unobjectionable for a condition, somewhat rare, which appears hitherto to have been unrecognised.* It is confined to late life, occurring in those over 40, and especially over 50. Its characteristic is simple weakness of the legs, with some slowness of movement, but without wasting, sensory disturbance, or reflex alteration. The knee-jerk is normal, and there is no foot-clonus. The malady develops very gradually and is slowly progressive, although it seems seldom to attain such a degree as to prevent standing. The condition of the legs resembles that in cases of paralysis agitans without tremor, in which the malady is manifested only by weakness, and stiff slowness of movement of the limbs, face, and trunk. Cases are met with in which the condition of the legs above described is associated with slight symptoms in the arms and face, such as characterise these cases of paralysis agitans. This senile paraplegia is probably a partial development of the morbid process of that disease; instead of being general, it is limited to the structures for the legs. If, as the condition seems to suggest, paralysis agitans depends on peculiar changes in the nutrition and function of the motor cells of the cortex cerebri (which impair the power and alter the action of the cells, but do not usually go on to such destructive alteration as entails secondary degeneration of the pyramidal fibres), we can readily conceive that such a process may be sometimes limited to the leg-centres, and may cause the symptoms. If so, the malady is a cerebral and not a spinal one, but it is mentioned here on account of its paraplegic features, and their resemblance to those of disease of the spinal cord. Degenerative changes may, however, be various in kind and degree, especially in late life; cases occur, similar to these, but with myotatic excess connecting them with the destructive changes of lateral sclerosis, while others present such symptoms as nystagmus or impeded articulation—evidence that the altered nutrition is wider in its range.

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* A note upon it by myself appeared in the 'Centralbl. f. Nervenkr.,' 1890.

CHRONIC SPINAL MUSCULAR ATROPHY

(PROGRESSIVE MUSCULAR ATROPHY; WASTING PALSY; AMYOTROPHIC LATERAL SCLEROSIS; CHRONIC POLIO-MYELITIS).

The disease which has long been known by the names "progressive muscular atrophy" and "wasting palsy" is characterised by slow wasting of the muscles, beginning in some one part, and usually spreading and increasing until it is wide in extent and extreme in degree. The changes in the muscles depend on changes in the spinal cord, a slow degeneration of the ganglion-cells of the anterior cornua, accompanied by a similar degeneration in the motor nerve-fibres which spring from the cells. With this degeneration of the cells and peripheral fibres there is usually also associated a degeneration of the pyramidal tracts in the cord, sometimes, at least, to be traced up to the motor cortex of the brain.

Chronic muscular atrophy was separated from primary paralysis by Sir Charles Bell, and was afterwards described more fully by Aran and Cruveilhier. Bell and Cruveilhier regarded it as a spinal disease. Aran (who first called it "progressive muscular atrophy") thought that it was primarily a disease of the muscles, a view that was afterwards held by Duchenne, and subsequently also by Friedreich. This opinion was supplemented by a theory that the wasting was due to a disturbance of the sympathetic. Lockhart Clarke first discovered the disease of the grey substance of the spinal cord, and Charcot specially associated the atrophy with the wasting of the ganglion-cells. When the constancy of the spinal lesion was demonstrated, it was thought that every form of chronic muscular atrophy was of spinal origin; but this view has proved erroneous, and it has been shown that there are forms of muscular atrophy which are purely local and idiopathic. The morbid process is not confined to the spinal cord. Atrophy of the nerve-cells from which the motor bulbar nerves arise often accompanies that of the spinal cells, and "progressive bulbar paralysis" is then associated with "progressive muscular atrophy:" it often comes on after the disease has existed for some time, and ends life; occasionally it initiates the malady.

Two varieties of the disease have been distinguished by Charcot, who has been followed in this by most subsequent writers. In the one variety the disease is manifested only by the wasting of the muscles; in the other there are, in parts other than those much atrophied, the indications of degeneration of the pyramidal tracts of the cord. In the latter cases, Charcot regarded the degeneration of the pyramidal tracts as the primary lesion, and the affection of the cornua as secondary. He therefore termed the cases of the first class "protopathic," and those of the second class "deuteropathic," and gave to the latter the name "amyotrophic lateral sclerosis." In Germany, however, and especially by Leyden, doubts have been expressed as to the validity of the distinction between the two classes of cases, and as to the sequence of the lesions in the second class. These doubts are not without foundation. The subject will be discussed when the pathology of the disease is considered, and reasons will then be given for the course here adopted of considering the two varieties together as essentially one disease.

But cases are also met with (although not frequently) that present every gradation to subacute polio-myelitis in one direction, and perhaps also to polyneuritis in another. Attempts are being made to classify these, but types may be multiplied indefinitely from the intermediate forms, and many more pathological and clinical observations are necessary before a useful classification of the cases can be made. In the following description the common forms are chiefly considered.

CAUSES.—The disease is more frequent in males than in females, the proportion being about three to one. It is chiefly a disease of adult life, commencing usually between twenty-five and forty-five. I have known it to begin at fourteen and as late as seventy, and a case is recorded in which it began at twelve;[*] there was wasting and rigidity in the limbs, tongue, lips, and lower face. But most cases of muscular atrophy in early life are idiopathic, and not spinal. Heredity is to be traced only in less than half the cases, and generally as an indirect neuropathic disposition. Rarely there is direct inheritance of the disease. Among instances that I have met with are a lady whose mother died from a similar atrophy, and another case (from which Fig. 135 is taken) in which a brother had died from some chronic disease of the cord, attended with wasting; but three cases with bulbar symptoms have been recorded, two of whom were cousins, the children of two sisters: the third, who had had syphilis, was a cousin, but his affection was untypical.[†] Direct inheritance seems to be most common in cases that occur late in life. When many members of a family suffer from

[*] H. Blumenthal, "Inaug. Diss.," 'Neur. Cent.,' 1884, p. 376.

[†] 'Virchow's Archiv,' 1889, p. 115.

muscular atrophy, the malady is nearly always idiopathic and not spinal. The affection occurs in all classes of society, and it is doubtful whether workers with the muscles furnish a larger proportion of the cases than can be accounted for by their greater exposure to certain exciting causes.

Of these more direct causes one of the most frequent is mental distress and anxiety, and this is especially met with in females and in late life. A severe fright has been thought to excite it. Another cause is exposure to wet cold, which is also a cause of many other chronic spinal diseases. Sometimes the exposure has been habitual; sometimes a single exposure has been effective, and some neuralgic pains, indicative of the deleterious influence on the nervous system, have followed the exposure and connected it with the later wasting. The pain has been either in the part afterwards wasted, or in some other, more common, seat of neuralgia. Thus one patient, after remaining in wet clothes for twelve hours, suffered for six weeks from severe neuralgia in the left side of the face, and then the muscles of the left shoulder began to waste. Although excessive use of individual muscles may cause them to waste, it is doubtful whether this influence produces general muscular atrophy. Injury to the cord, such as results from concussion of the spine, is a rare cause. It more often produces disseminated myelitis, which may be manifested by muscular wasting combined with other symptoms.* In a few instances I have known progressive atrophy, of typical characters, to slowly follow a concussion, as if this had set up a perversion of the nutrition of the nerve-elements. Still more rarely a fall, injuring one limb, has been followed by muscular atrophy commencing in this limb and becoming general. Thus a woman fell downstairs, and pitched on her left hand and wrist; she had pain in the arm for a long time; two years after the fall this arm began to waste, and the atrophy ultimately became general. The relation might be passed as an accidental coincidence, were it not that in other central diseases—even, for instance, in paralysis agitans—the symptoms may commence in an injured limb. In rare cases, the disease develops, during adult life, in a subject of old infantile palsy (polio-myelitis), and may start from the most affected part (see p. 364).

The disease sometimes succeeds syphilis, and no other cause may be traceable. As with other degenerative diseases, an interval of years elapses between the primary disease and its nervous sequel. The cases that I have seen after syphilis have been typical in course, and evidently degenerative in nature. That syphilis has some share in the causation of these cases is probable from its relation to other diseases, such as tabes; but it is noteworthy that in one case the wasting commenced during, and in another directly after, an energetic mercurial course. This relation is intelligible if the disease is due to a product of the syphilitic virus, and not to the organisms themselves (see Tabes). Occasionally syphilis and a neurotic heredity can both be traced. In the cases in which atrophy has followed an acute specific disease, especially measles, and has begun in early life, it is probable that the malady is a chronic neuritis and not the central affection now under consideration. Such cases are known as of the "Peroneal type," from the muscles in which wasting is first obtrusive, and are separately described at a later page. General muscular atrophy may result from lead poisoning, but this form is not, as a rule, progressive in character when its cause has ceased to act. It resembles the ordinary form of progressive muscular atrophy, however, in seat and features, and thus differs from the common atrophic palsy of the extensors that is produced by lead. In many cases of progressive muscular atrophy no cause for the disease can be traced. When obtrusive symptoms have followed some adequate cause, it is not uncommon for this to have been preceded by slight symptoms, which show that the malady already existed. A careful inquiry for such symptoms should never be neglected.

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* In one unusual case the wasting (ultimately general) began in the deltoids a month after a fall, which probably fractured the base of the skull (Manor, 'Alienist and Neurologist,' 1886, p. 430).

SYMPTOMS.—The definite symptoms of the disease are often preceded by aching pain in the part afterwards wasted, rarely severe. Sometimes there is such pain in the spine or elsewhere, especially, as already stated, in the cases that are due to exposure to cold. Rarely some sensation other than pain precedes the local symptoms. Thus in one case a sense of coldness preceded the atrophy in each limb that was attacked. Weakness and wasting usually come on together, but either may first attract the attention of the patient. In the shoulder and back, the loss of power is usually first noticed, and in such covered parts the wasting may become considerable before it is observed. In the hand the wasting is often first noticed, but sometimes it is the impairment of some delicate action, such as writing, that draws attention to the part. The affected muscles lose their proper shape, and there is flattening, or even a depression, where there should be a prominence. If a patient is fat, however, the wasting may cause at first very slight alteration in the external aspect.

The disease commences in the arm in nine tenths of the cases, and as frequently in one arm as in the other. It begins with almost equal frequency in the hand and in the shoulder muscles. From the part first affected the disease spreads to other parts of the same limb. Before it has attained a considerable degree in one limb, it usually shows itself in the corresponding limb on the other side; often in the muscles corresponding to those in which it commenced, sometimes in those affected second in order of time. As the muscles waste, paralysis results of various character and degree, corresponding to the atrophy. In the hand, the thenar muscles and interossei are usually the first to suffer. The thenar eminence becomes flattened, and the base of the first metacarpal bone becomes prominent. Of the interossei, the atrophy of the abductor indicis is especially conspicuous; the normal prominence gives place to a hollow beside the metacarpal bone when the thumb is abducted. Depressions form between the metacarpal bones on the back of the hand, and also between the flexor tendons in the palm in consequence of the wasting of the lumbricales. The forearm muscles may be next involved, the flexors usually before the extensors; and with the flexors the supinators may suffer, or they may escape until the biceps is involved. Occasionally the disease begins in the forearm, and then especially in the extensor muscles of the fingers, sometimes in those of the thumb, especially of its phalanges. The several parts of the long finger muscles may suffer unequally; the ulnar extensors usually suffer most. Of the shoulder muscles, the deltoid is generally the first to manifest the disease, and in some cases which begin in the hand the deltoid suffers before the forearm muscles. The rounded contour of the shoulder becomes changed (Figs. 7, 134, 135), and the head of the humerus can be recognised beneath the acromion. It is not rare for part of the del-

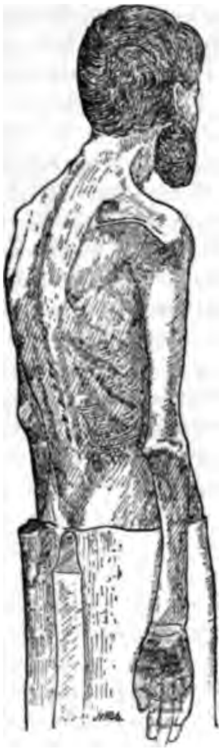

FIG. 134.—Progressive muscular atrophy. Wasting of the muscles of the back and arms; in the forearm scarcely any muscular tissue can be recognised, and in the hand all the muscular prominences have vanished. (Drawn by Dr. H. R. Spencer.)

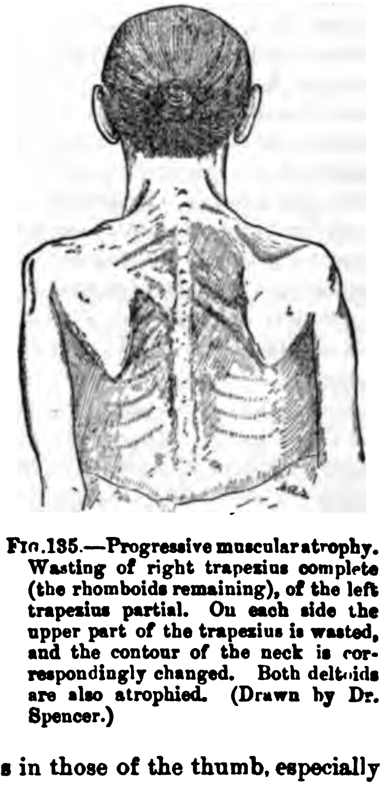

FIG. 135.—Progressive muscular atrophy. Wasting of right trapezius complete (the rhomboids remaining), of the left trapezius partial. On each side the upper part of the trapezius is wasted, and the contour of the neck is correspondingly changed. Both deltoids are also atrophied. (Drawn by Dr. Spencer.)

toid to suffer and part escape: we have seen (p. 30) that the several portions have different functions and associations, and this probably involves a relation to separate groups of nerve-cells. The wasting of the deltoid is soon followed by that of the other muscles of the upper arm and of the scapula. The triceps usually suffers less and later than the biceps, but sometimes the reverse is the case. The supra- and infra-spinati are often affected with the deltoid. In exceptional instances the deltoid or hand muscles escape, and there are cases (perhaps a special group) in which the upper arm and shoulder muscles waste even to an extreme degree, and the forearm and hand muscles escape entirely.

In most cases, the wasting early involves the muscles of the back, and it sometimes begins in them. The middle and lower parts of the trapezius usually suffer first; the rhomboids and erectors of the spine at a later date. The affection of the trapezius is readily recognised if the patient tries to put the shoulder back (Fig. 135). The highest part of the trapezius presents a remarkable indisposition to atrophy; it often remains intact to the last, and then may contrast with the wasting below it, standing out on each side, like a cord passing from the occiput to the shoulder. Hence Duchenne termed this part the *ultimum moriens*. I have, however, seen several otherwise typical cases (beginning either in the deltoids or in the hands) in which the highest parts of the trapezii suffered before the middle parts. The levator anguli scapulæ also generally escapes, even when all the muscles about it are wasted. The serratus, latissimus, and pectoralis major are usually affected later. They may escape wholly or in part; isolated bundles of the pectoralis may waste, the intervening parts escaping, or either the upper or lower part of the muscle may atrophy alone. According to the affection of the muscles that are attached to the scapula, the position of the bone changes, and it becomes rotated under the influence of the muscles that are unaffected and unopposed (Fig. 7, p. 28). The muscles that extend the head on the spine often suffer in considerable degree, and from this there results a difficulty in the carriage of the head (Fig. 136). It is habitually inclined backwards, so as to balance it on the spine with but little muscular exertion; if moved forwards, it falls so that the chin touches the

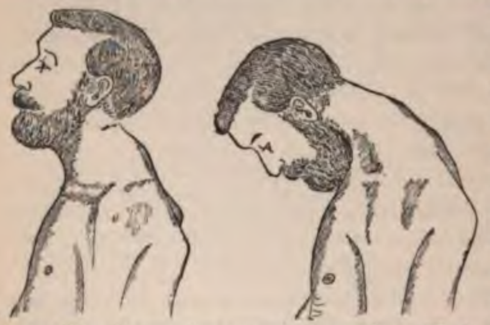

FIG. 136.—Progressive muscular atrophy. Weakness of the muscles of the neck. A, habitual posture of the head inclined backwards. B, position into which it falls if the patient attempts to keep it in the normal balance. (Drawn by Dr. Spencer.)

chest. It can be brought back into its former position only with difficulty; the patient has to incline the trunk backwards, so as to bring the head nearly into the vertical position, and then, with a sudden contraction of the sterno-mastoids, and a jerk, the head goes back into its former posture. The increased innervation of the weak extensors of the head often causes a synergic over-action of the frontalis muscles, which are normally associated with the extensors (so that the eyebrows are raised when the head is put back to look upwards). The skin at the back of the neck lies in transverse folds when the neck is extended. The patient may be unable, when lying in bed, to move the head from side to side. The sterno-mastoids also are often wasted; either the sternal or clavicular part may be most affected. In striking contrast to the general wasting of the neck is the condition of the platysma myoides, which always escapes, and may become hypertrophied in a vain attempt at compensation.

The muscles of respiration suffer in the majority of cases, and their impairment constitutes a grave source of danger to life. The intercostals rarely escape altogether; the diaphragm is involved in many cases, and respiration is then carried on by the intercostals and superior thoracic muscles. On the other hand, the intercostals may suffer much and the diaphragm remain free; respiration is then purely abdominal, and the walls of the thorax may be almost motionless in breathing, or there may be merely a slight movement of elevation of the upper ribs. The chest becomes flattened in front, and narrow from before back, from the influence of atmospheric pressure on the ribs, unopposed by the intercostals. Either the upper or lower intercostals may suffer most. When the diaphragm is paralysed, the upper chest muscles often suffer more than the lower, and a compensatory increased movement of the lower ribs carries forward the abdominal wall, and may suggest that the diaphragm is acting when it is not. A careful examination will always prevent the error. The muscles of the abdominal wall occasionally waste, but far less frequently than those of the thorax.

Wasting in the legs is much less common than in the arms, and if it occurs is usually slighter in degree; but occasionally the disease first manifests itself in the legs, and is more intense in them than elsewhere. The glutei, extensors of the knee, and the muscles in the front and on the outer side of the lower leg, are those that are most affected. We shall presently see, however, that the legs are often paralysed when they are not wasted, and sometimes they are the seat of wasting which differs in certain features from that which is the special characteristic of the disease.

The face almost always escapes the general atrophy, and its normal appearance may present a striking contrast to the rest of the body. In many cases, however, the lips are paralysed as part of the bulbar palsy that so often accompanies the spinal disease. A remarkable case in which the face shared the atrophy of the limbs, and the tongue

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escaped, has been recorded by Langer;[*] on the other hand, the tongue has been the first part to suffer, the arms being affected a month later.[†]

As the wasting progresses, the appearance of the parts in which it is most advanced becomes extremely changed. All trace of muscle may disappear from parts of limbs or even from an entire limb (Fig. 134); and there is usually a wasting of the adipose tissue as well as of the muscle, so that the bone seems to be covered only by fascia and skin. The transverse processes of the vertebræ may be felt in the hollow beside the spine, and the bony prominences about the shoulder may be almost as conspicuous as in a skeleton, so that beneath the acromion there may be a groove, into which the finger can be placed, in consequence of the descent of the head of the humerus from the glenoid cavity. The unequal affection of antagonistic muscles leads to various contractions and deformities. In the hand, especially, distortion is apt to occur; from preponderant paralysis of the interossei and contraction of the long flexor and extensor muscles, the "claw-like hand" develops in extreme degree (Figs. 19 and 20, p. 36). If all the muscles of a part are equally and simultaneously affected, no deformity results unless an unsupported part yields to the influence of gravitation (Fig. 134). Lordosis is very common in cases in which the trunk and hip muscles are involved. It is often the indirect result of the weakness of the extensors of the hip-joint, in consequence of which the pelvis is unduly inclined forwards, and carries with it the lower lumbar vertebræ; the upper part of the trunk has then to be inclined backwards, to bring the centre of gravity over the base of support; it disappears when the patient sits, and the pelvis, resting on the ischial tuberosities, is no longer inclined. This was the case, for example, in a woman with extreme lordosis from progressive muscular atrophy, in whom, when standing, a plumb-line from the seventh cervical spine fell three inches from the sacrum and an inch and a half from the buttock. Sitting, her spine was perfectly straight.

The electrical irritability of the wasted muscles presents changes which vary in character in different cases. When the wasting is slow, there is usually a diminution in both faradic and voltaic irritability, similar in character to each current. The irritability fails with the muscular nutrition, and when the wasting is great only a slight contraction can be obtained, even with a strong current. When the wasting is extreme, irritability at last becomes extinct, but the voltaic irritability of the muscular fibres persists long after the faradic irritability has disappeared. It may be normal in degree, or lowered, but is seldom increased in such cases. The quality of the voltaic irritability may also be normal, but sometimes ACC (anodal closure contraction) occurs as readily, or more readily than

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[*] Vienna Medical Society, March 17th, 1882.

[†] Birdsall, 'Journal of Nerv. and Ment. Dis.,' 1887, xiv, p. 256.

KCC. Tetanic contraction during the passage of the current is produced with undue readiness compared with the closure contractions, and opening contractions are often caused by currents but little stronger than those that cause closure contractions. Thus there may be the qualitative change of the reaction of degeneration without the voltaic increase (see p. 56). When there is the rapid and considerable paralysis described on p. 483, either at the onset or during the course of the disease, the palsy being followed by rapid wasting, there may be quick extinction of faradic with actual exaltation of voltaic irritability, and the reaction of degeneration may be present in all its characteristics. Between these forms every intermediate condition may be met with, including the mixed reaction described on p. 26.

The mechanical irritability of the muscles is considerably increased; a tap causes a local contraction of the fibres struck. Moreover, spontaneous flickering contractions of parts of the muscles are very common, now of one bundle, now of another, conspicuous to the eye, although scarcely felt by the patient. This "fibrillation," as it is termed, is so frequent as to be characteristic, but it is not invariable, nor is it confined to this disease. It may sometimes be observed in muscles that are not yet invaded by the wasting, but where it is observed atrophy usually follows.

In the parts affected by the characteristic wasting, all reflex action is abolished, clearly in consequence of the damage to the motor part of the reflex arc; the afferent portion of the arc is unaffected, for, as we shall see, there is no loss of sensation. The myotatic irritability ("tendon-reflex action") is also lost, and lost early. The knee-jerk, for instance, disappears as soon as there is even a slight diminution in the bulk of the thigh muscles, in cases in which the legs are the seat of primary wasting. The muscles are flaccid and toneless—a condition that may be conveniently termed "atonic atrophy." To this common rule, however, exceptions are sometimes met with; there may be from the first rigidity of the affected muscles. When this is the case, the wasting does not go on to the entire destruction of the muscle; it is often trifling, but may be considerable. With this rigidity there is a preservation of myotatic irritability. We may term this, "tonic atrophy." It is generally associated with spastic paralysis, without wasting, in some other part. For instance, in one case the forearms and hands were the seat of this tonic atrophy, and in the legs there was simple spastic paraplegia. We shall presently consider the pathology of this condition.*

Sensory symptoms are usually slight and always subordinate. They are confined to pain, usually dull and rheumatoid in character, felt chiefly in the limbs in which the malady is most active. Such pains are common at the onset, as we have already seen, and they may recur from time to time during the course of the disease. Sometimes vague feelings of "numbness" or "deadness" are complained of, but cutaneous sensibility is never impaired. (When anæsthesia accompanies muscular wasting, both symptoms are due either to chronic meningitis, damaging the nerve-roots, or to disseminated focal myelitis or multiple neuritis.) Nor do the muscles lose such sensibility as they normally possess. Perception of posture persists, and the muscles are not only sensitive to pressure, but are often more sensitive than in health both to pressure and to extension.

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* It should be noted, however, that, in the muscles that are the seat of toneless wasting, when the atrophy has attained an extreme degree, slight rigidity may develop, accompanied by considerable tenderness of the muscle. It is probable that this rigidity is idiopathic, and due to the structural changes in the muscles presently to be described. This condition, which should be distinguished from the initial rigidity, is rare: the early relaxation is usually maintained to the last.

When the arms are the seat of such atrophy as has been described, the legs, if not also wasted, may be normal, or they may be slightly affected, the thighs thin, and the knee-jerks lost. Often they are the seat of gradual loss of power without wasting, and with an increase of myotatic irritability. The knee-jerk is excessive; a clonus can readily be obtained, and the reflex over-action of the muscles may increase to actual rigidity and spasm, so that there is the condition of spastic paraplegia described in a preceding chapter. It is rare, however, for the spasm to reach the higher degree of intensity, and for the muscles to present the massive firmness that characterises the simple form of spastic paraplegia. On the other hand, there is often some diminution in the size of the muscles, and thus we have a gradation to the condition of tonic atrophy just described. In this condition, moreover, there is usually only a moderate diminution in electrical excitability, which does not go on to extinction. The one condition does not seem to pass into the other; the state of rigidity and myotatic excess does not give place to muscular relaxation; atonic atrophy does not succeed the tonic wasting. In very rare cases, of which I have seen a few instances, there is a similar condition in the hands when the shoulders are the seat of simple atonic atrophy. The forearms are then rigid, moderately wasted, with myotatic excess, while the muscles of the shoulders are extremely wasted and absolutely flaccid. In still more rare cases the whole arms are thus affected, are thin and rigid, and in no part is there atonic atrophy. In one remarkable case of this character, the muscles of the trunk participated in the spasm; after coughing or yawning, respiration would be checked for a few seconds by general spasm, and whenever the patient was raised from the bed, the arms, legs, back, and neck became stiff in tetanoid rigidity, the head being bent backwards.

The combinations of the ordinary atonic wasting and loss of power, without wasting and with increased myotatic irritability, are exceedingly numerous and variable. The most frequent, as already stated, is flaccid atrophy in the arms and spastic paralysis in the legs; but I have twice seen atonic atrophy below the knees, with weak but well-nourished thigh muscles and much increased knee-jerk. The variable combination of these conditions gives rise to differences that are more apparent than real, and may easily be allowed undue weight in classification.

The extreme emaciation of the most affected parts shows that the adipose tissue wastes as well as the muscles. The atrophied limbs are usually cold, and may be livid or pale, but there is no tendency to acute vaso-motor disturbance in the ordinary form of the disease. I have known the skin of the face to become thin and smooth, so that, in one instance, the dark iris could be seen through the closed eyelids, but such a change is quite exceptional. In another patient there was a very remarkable form of local atrophy. Certain areas of muscles underwent wasting, the rest being normal; the affection commenced by a livid discoloration of the skin, and the wasting seemed to involve the skin, subcutaneous tissue, and muscle, causing local depressions. Considerable tracts of the trapezii were thus affected, and smaller spots in the arms and legs. The patient was a single woman of thirty-five, and the disease, which did not show a strongly progressive tendency, is probably a special affection, perhaps of local nature.

The functions of the sympathetic are not, as a rule, deranged. Dilatation or contraction of one pupil has been frequently observed, chiefly in association with atrophy of the muscles that are supplied from the lower part of the cervical region; and it no doubt depends on the disease of the spinal cord, and not of the sympathetic itself. The reflex action of the iris is usually normal, and optic nerve atrophy never occurs.* Nystagmus is present in rare cases.

The visceral functions are usually little disturbed. Sexual power is often lost. The sphincters rarely suffer, even when the wasting is general and extreme; occasionally they are involved, and they may even suffer early. In other cases, in which the legs become weak after the wasting sets in in the arms, the affection of the sphincters may coincide with that of the legs. In the composition of the urine, slight changes have been found, but not constantly. Urea has been increased in some cases, lessened in others.† The quantity of lime has been found to be increased (Fromman), that of kreatin diminished, even to one tenth of the normal (Rosenthal, Langer). Glycosuria has been observed in association with bulbar symptoms.* The lungs may suffer from the impairment of breathing power, when the intercostals and diaphragm are weakened.

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* I have once met with a remarkable reflex fixation of the eyeballs in a case of advanced progressive muscular atrophy. If the patient, looking to one side, was suddenly told to look at an object on the other side, his head was instantly turned towards the second object, while the eyes remained fixed on the first, by a movement corresponding to that of the head but in the opposite direction, and then, after a few seconds, they were slowly moved towards the second object. The phenomenon continued to the end of the patient's life. As I pointed out in an account of this curious condition ('Brain,' vol. i), it is interesting evidence of a normal reflex mechanism in the fixation of the eyes; this was, as it were, isolated by disease, which lessened voluntary control over it.

† In a patient who weighed 7 st. 13 lbs., and whose urine varied between 760 and 900 cubic centimetres, I found the daily excretion of urea to vary between 10·7 and 15 grammes, the average being 13 grammes. This is just half the normal average for a man of that weight, which would be (according to Parkes), 24 grammes.

Varieties.—The chief varieties of the disease depend on the relative distribution of the three conditions: (1) atonic atrophy, becoming extreme; (2) muscular weakness with spasm, but without wasting, or with only slight wasting; and (3) tonic atrophy, rarely extreme in degree, with myotatic excess. The commonest condition is to have atonic atrophy in the arms and upper part of the trunk, with simple weakness and spasm in the legs. Atonic atrophy in both arms and legs is less common, and the least common is tonic atrophy alone, in the arms, or universal. The last is, indeed, extremely rare. It is important to note, however, that these conditions may coexist in every degree and combination; between universal atonic atrophy on the one hand, and universal spastic paralysis without wasting on the other, there is every gradation. The latter does not come into the category now under consideration, but similar cases are met with in which there is atrophy of a few muscles (as, for instance, of the hands only), which complete the series. The varieties due to the different combinations of atrophy and paralysis have been already alluded to.

A peculiar form of muscular atrophy has been mentioned, first conspicuous in the peroneal regions and spreading thence to other muscles of the leg and to the hands. It is characterised by occurrence in early life, and often in several members of the same family, and has been termed the "peroneal type" by Dr. H. Tooth. It is probably not dependent on the spinal cord, and is considered more fully at a subsequent page.

Complications.—Progressive muscular atrophy is occasionally accompanied by the symptoms of some other degenerative disease of the spinal cord. The paralysis with spasm, already described, can scarcely be regarded as a complication; it is rather part of the disease, and its relation to the other symptoms will be considered when we discuss the pathology of the affection. In rare cases, muscular atrophy in the arms is accompanied by the symptoms of locomotor ataxy in the legs. By far the most frequent complication is bulbar paralysis, weakness of the lips, tongue, pharynx, and often of the laryngeal muscles. It is the expression of a degenerative process in the medulla, similar to that which, in the spinal cord, causes the affection of the limbs. It may come on at any stage of the disease, may precede the spinal symptoms, or only come on when these have attained a considerable degree. In characters, the bulbar palsy may resemble perfectly that which occurs in isolated form, and will be described among the diseases of the brain. Frequently, however, there is but little paralysis of the tongue, even when swallowing and articulation are much impaired. There may also be slight interference with articulation when there is no marked bulbar palsy. A minor complication is the neuralgic pain, which, as already stated, is sometimes troublesome in the early stage of cases that are due to cold. In several patients I have known headache to be frequent and severe throughout the course of the disease. General paralysis of the insane has been met with as an exceptional complication.*

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* Rovighi and Melotti, 'Riv. Sper.,' 1888, p. 815.

Course.—The malady, in most cases, is steadily progressive, as its name implies, but in the rate of its progress it varies in different cases, and in the same case at different periods. It may, moreover, become stationary (sometimes as the result of treatment) at any period in its course, and when once actually arrested, it does not usually again become active. Unfortunately, the tendency to cessation is greatest in the later stages of the disease, when there is little except life to be preserved. Sometimes progress ceases at an earlier stage, and chiefly, I think, in those cases in which the atrophy is strictly symmetrical, and develops simultaneously, or almost simultaneously, on the two sides. Thus I have met with several cases in which there was symmetrical wasting in the two hands, or in certain muscles of the two forearms or of the two upper arms, and in which the atrophy, after attaining a considerable degree in its limited seat, had become stationary and continued so. Occasionally some symptom (as weakness of the sphincters) may pass away, although the atrophy progresses.

When the progress at the commencement is rapid, it usually continues rapid, until the disease has attained a wide extent, although the acute local onset mentioned below may be followed by slow extension. When it begins slowly, it is usually slow throughout.

Although the disease sometimes commences in the second arm very soon after its onset, it more commonly makes some progress in its primary seat before beginning on the second side, and the interval that elapses varies with the rate of extension. It often happens that a year intervenes between the affection of the two arms, and I have known, in a very chronic case, the atrophy to slowly progress in one arm for seven years before it showed itself in the other. It is not common for the arms to be reduced to practical helplessness in less than two or three years, but the hands may become useless in as short a time as six months, while in one case the wasting, commencing at the shoulder, had invaded the whole arm in the course of a month. The shortest time in which I have known a patient to reach the last stage of the disease is nine months.

With any rate of general progress, the otherwise uniform course of the disease may be broken by the occurrence of almost sudden palsy in a certain group of muscles. Considerable loss of power, it may be absolute paralysis, comes on in a few days, or even in a few hours. It may occur at the onset of the affection. The extensors of the wrist and fingers are the muscles most commonly thus affected. The weakness is usually followed by a well-marked degenerative reaction in the muscles. I have seen several cases which began by such subacute or even acute paralysis of the extensor muscles, first in one arm and soon afterwards in the other; the initial condition closely resembled the paralysis from lead-poisoning, even to the escape of the supinator longus and ext. ossis met. pol. This cause was, however, excluded, and soon the muscles of the shoulder and back presented commencing slow progressive atrophy, followed by wasting of the interossei until a typical state was reached. In another case, the quick loss of power was confined to the extensors in one arm, which were already weak and slightly wasted, most of the other muscles of the upper limbs having been long atrophied. When there is weakness of the legs, without wasting, the onset of this may coincide with the atrophy of the arms, or may succeed it at any interval. In one case five years elapsed, after the arms began to waste, before the legs became weak. It is very rare for the paraplegic weakness to occur first.

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* Tambroni, 'Riv. Sper. de Fren., &c.,' 1887, xiii, p. 184.

The chief danger to life is from pulmonary maladies, rendered grave from the weakness of the muscles of respiration. The common complication of bulbar paralysis is another frequent cause of death, either by the interference with swallowing and nutrition, or by the laryngeal paralysis. Less commonly, death results from bedsores and septicæmia, or from intercurrent maladies.

Pathological Anatomy.—The wasting of the muscles is as evident after death as during life. They are reduced in size and pale in colour. Sometimes there is little in the tint of what remains to suggest muscular tissue. Parts of a muscle may be hardly distinguishable from adjacent fat. On the other hand, the bulk of the muscle may be dark, and pale streaks in it may mark the position of local degeneration. Under the microscope, the fibres present various changes, and of these four are well defined. (1) There may be simple narrowing of the fibres, without any considerable change in their striation (Fig. 137), although the striæ often seem to be further apart than normal, and sometimes the fibrillary segmentation is unusually distinct. (2) Simple fatty degeneration, in which the transverse striation gives place to a granular appearance (Fig. 138), the granules becoming larger and fewer (Fig. 137) until ultimately distinct globules are scattered through the sheath. Where the muscle resembles fatty tissue to the naked eye, the microscope may show only sarcolemma sheaths containing groups of globules. (3) Muscular fibres are seen in which the sheath contains only a clear material enclosing a few fatty globules, and a few transverse striæ, faint, as if fading away. It is probable that this is not the result of fatty degeneration, but of a different process, which has been termed "vitreous degeneration,"—a sort of dissolution of the striæ, indicated by the appearance of such fibres as are shown in Figs. 137, 139, 140. (4) A longitudinal striation develops in the fibre, and at first coexists with the transverse striation; but ultimately the latter becomes indistinct, and the fibre looks like a fasciculus of longitudinal connective-tissue fibres (Fig. 140). Sometimes, with the longitudinal striation, the fibre presents a transverse striation very much finer than normal, the striæ being narrower and nearer together, as if from a division of the "sarcous elements." This change may sometimes be seen alone, and may be present in only one part of a fasciculus. Thus at one place the number of striæ in $\frac{1}{1000}$ inch was only seven, while in another region of the same fasciculus seventeen were to be counted in the same space. A tendency to transverse fissuring has also been described, but this is probably artificial. Fatty globules accumulate between the fibres (Fig. 140), accompanied in some cases with granules and masses

FIG. 137.

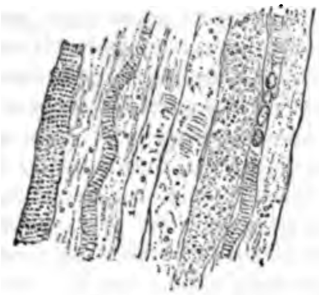
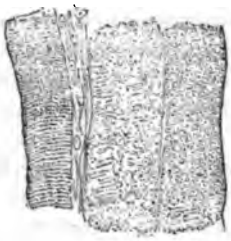


FIG. 138.



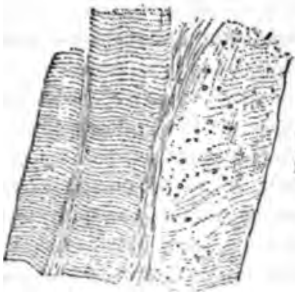


FIG. 139.

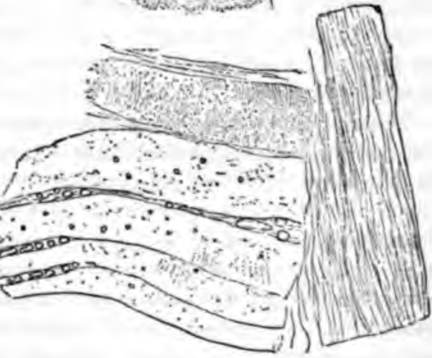


FIG. 140.

FIBRES OF WASTED MUSCLES IN PROGRESSIVE MUSCULAR ATROPHY.

FIG. 137.—Narrowed fibres with nearly normal striation; others clear, containing a few fat globules, and a few faint transverse striæ, and some longitudinal striation: large globules of fat lie in the interstitial tissue in front of one narrowed fibre.

FIG. 138.—Granular degeneration: a fibre with normal transverse striation presents also indications of longitudinal striation. Increase of nuclei of interstitial tissue.

FIG. 139.—Two normal fibres and one presenting the clear homogeneous aspect, with a few fat globules and some faint striation.

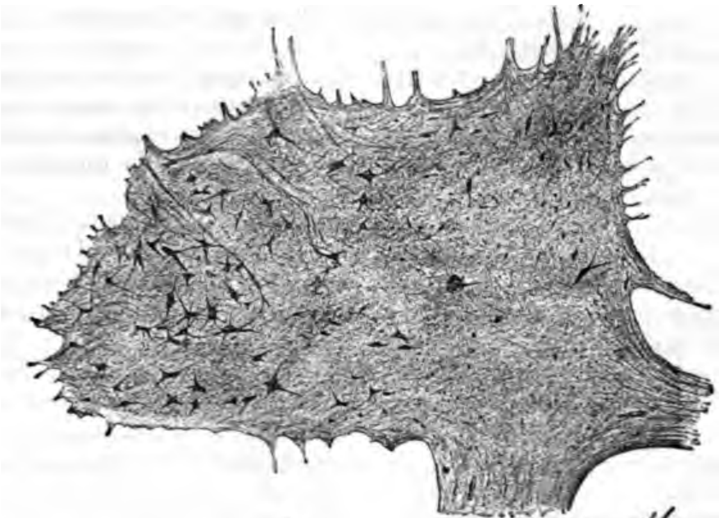
FIG. 140.—Several fibres similar to that of the last figure, with globules of fat between them. On the right is a fibre which has undergone complete longitudinal striation, the normal striæ having disappeared, so that it resembles the adjacent interstitial fibrous tissue. Other fibres in the same muscle were in intermediate stages of degeneration, some transverse striation coexisting with the longitudinal striation.

of reddish-brown pigment. There is often also an increase of the nuclei (Fig. 188), and sometimes of the fibres of the interstitial tissue. Two or three rows of nuclei may lie between the fibres. The capillaries may be dilated and distended. It is very common to see muscular fibres that are much altered side by side with others that present a nearly normal appearance. Ultimately the sheaths become empty, and shrink, and may be scarcely distinguishable from the interstitial fibrous tissue.

The peripheral nerves contain many degenerated nerve-fibres, and the terminal branches for the muscles a still larger number. If the nerves are traced up to the cord it is found that the degenerated fibres come only from the anterior roots. These are conspicuously changed to the naked eye, small and grey. They may resemble fine threads of connective tissue, and under the microscope no nerve-fibres may be found in them, or only a few may remain of normal aspect, the rest being in various stages of degeneration, or represented only by their empty sheaths. The degree of affection of the anterior roots corresponds to the wasting in the parts supplied by them. The posterior roots are normal.

The spinal cord is often softer than natural at the affected part, and the white substance of the lateral columns may be grey and translucent in aspect, especially in the cervical enlargement. Under the microscope morbid changes are seen in the anterior cornua, and also, in most cases, perhaps in all, in the antero-lateral white columns. The change in the anterior cornua corresponds in its intensity to the origin of the nerves to the most affected parts, and since the latter are usually the arms, the disease is generally most intense in the cervical enlargement. In stained sections the grey matter of the horn is less deeply tinted than normal, but in some parts it may stain more deeply, especially in the circumference of the cornu and the processes of grey matter which project into the white column. Occasionally, larger areas have a dense aspect and stain deeply, but the central part of the cornu is generally pale and wasted in aspect. Most of the large nerve-cells have disappeared; many entirely, while others are represented only by small angular bodies. Not a single large cell may be seen in a section which, in a normal cord, would contain a considerable number. Frequently, however, a few large cells can still be seen, but most of these have lost their processes and are more globular than normal. The interstitial tissue is also changed. The nerve fibrillæ waste with the cells, and there is an increase of the small, angular, and stellate cells and other connective-tissue elements. The larger vessels are dilated and surrounded by unduly wide spaces, but there is no considerable distension of the capillaries. The cornu as a whole is not usually changed either in size or shape. Similar alterations may be traced through the dorsal region. In the lumbar enlargement, the grey matter may be normal, even when it is much altered in the cervical region; but if the legs are wasted, there are changes similar to those already described. When the atrophy begins in the legs, the disease may be more intense in the lumbar than in the cervical enlargement; but more often any degeneration that exists is slighter, and a larger number of normal or slightly changed cells can be seen. Sometimes certain groups of cells are little affected while others are much atrophied. The degeneration may involve to some extent the intermediate grey matter between the cornua, but here it ceases; the posterior horn is always normal.

FIG. 141.



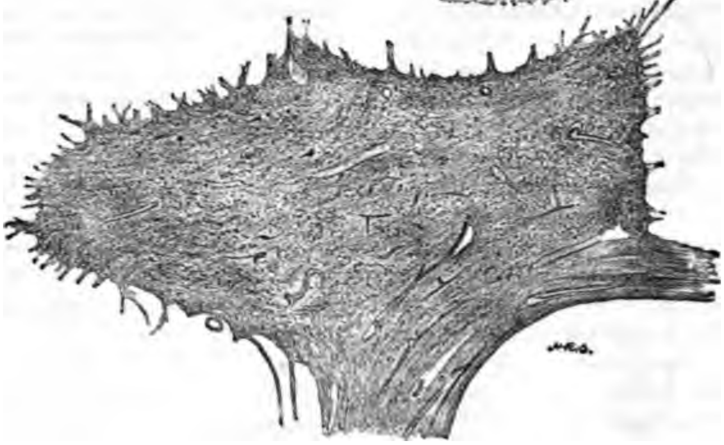


FIG. 142.

FIG. 141.—A normal anterior cornu for comparison with the next figure.
FIG. 142.—Progressive muscular atrophy; anterior cornu, cervical region.
(From drawings by Dr. H. R. Spencer.)

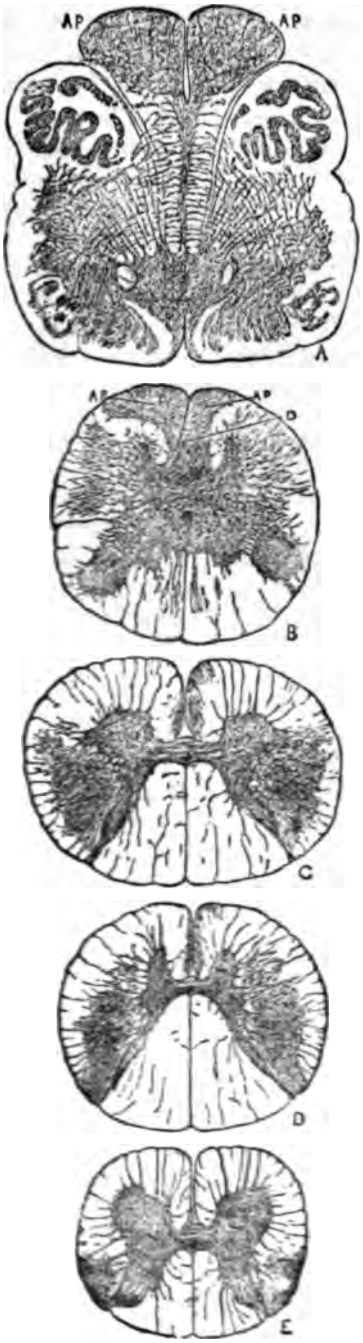


FIG. 143.—Progressive muscular atrophy; degeneration of the anterior cornua and pyramidal tracts. A, medulla oblongata, complete degeneration of the anterior pyramids A P; B, at the upper part of the decussation of the pyramids; D, the decussation of the degenerated fibres; A P, the pyramids, still incomplete. C, cervical, D, dorsal, E, lumbar sections. The degeneration of the anterior cornua is complete in C, but in E a few cells remain, for the most part without processes. In C and D the degeneration of both anterior and lateral pyramidal tracts is conspicuous; in E the anterior tract has ceased, and the lateral tract extends up to the surface of the cord, from which it is separated in C and D by the undegenerated direct cerebellar tract. In C and D the degeneration extends forwards in front of the lateral pyramidal tract, but gradually ceases. The fibres of the anterior commissure are also degenerated.*

There is distinct degeneration of the anterior root-fibres passing from the cornu through the anterior column. A few fibres may remain, but whole fasciculi appear to be replaced by fibrous tissue. There is also degeneration of the fibres of the anterior commissure, in consequence of which it stains much more deeply than normal, in the regions in which the grey matter is considerably diseased.

In the white columns there is usually considerable, and often almost complete, degeneration of the pyramidal tracts, anterior and lateral.[*] The area of sclerosis, resulting from the degeneration of the two tracts, varies in extent according to the size of the anterior tract, and to the distance which it extends down the cord. In Fig. 144 this is illustrated in an unusual manner, in consequence of an inequality in the decussation of the pyramids at the medulla, fewer fibres of

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* In this case the arms were greatly wasted and flaccid; the legs were paralysed, rigid, with moderate wasting. The muscles of the back were also atrophied. In the arms the weakness and wasting came on at the same time, in the right arm some time before the left. The shoulder-muscles suffered first, but the atrophy quickly spread, and was ultimately extreme in the muscles of the shoulders, forearms, and hands. The disease ran a rapid course; death occurred two years after the gradual onset.

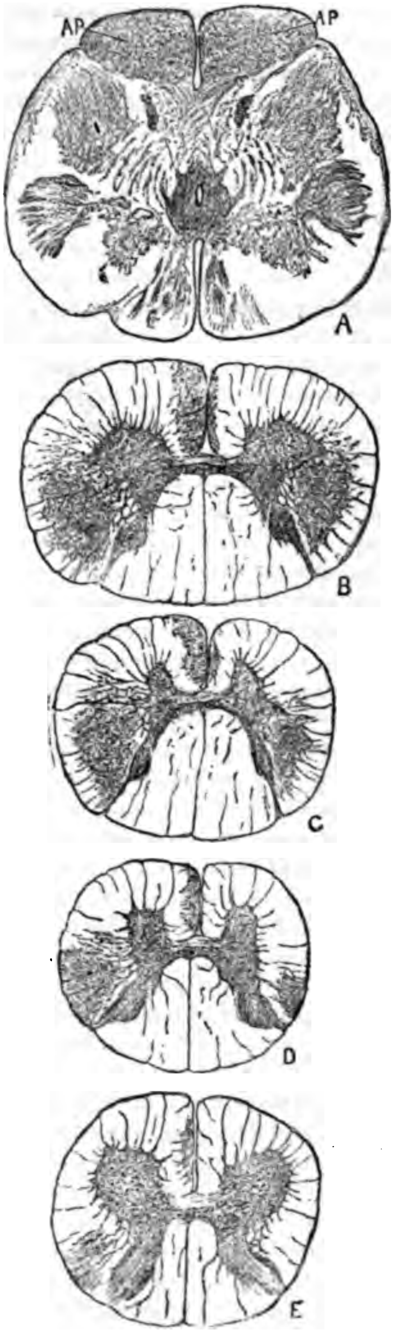

FIG. 144.—Progressive muscular atrophy. A, medulla oblongata; degeneration of the anterior pyramids A P. B, cervical, C, first dorsal, D, first lumbar, E, mid-lumbar sections. The nerve-cells have disappeared from the anterior cornua, except in E, in which a few remain. There is complete degeneration of the pyramidal tracts, anterior and lateral. The decussation at the medulla was unequal in this case; of the left pyramid fewer fibres crossed than of the right, and hence the left anterior pyramidal tract is larger, while the right lateral tract is smaller than normal, and, moreover, has ceased at the middle of the lumbar enlargement, to which the left anterior tract extends.[†]

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[*] I have not yet met with a single case of progressive muscular atrophy in which the pyramidal tracts were unaffected. Such cases are extremely rare, but one has been published by Strümpell ('Zenker's Zeitsch.,' 1887, and 'Neur. Centralbl.,' 1888, p. 139) which was characterised by the general wasting of the arm, a very slow course, and the absence of leg symptoms. The spinal nerve-cells and motor nerves were degenerated. Strümpell suggests that the affection may have begun in the peripheral motor nerves, and have ascended these to the cord. If so, the case belongs to a separate class.

[†] The patient was a man forty-eight years of age. The disease followed a severe exposure to cold. The wasting of the arms was extreme, and they were absolutely powerless. The legs were much less wasted than the arms, but very weak, the loss of power being out of proportion to the wasting.

the left pyramid having crossed than of the right, so that the left half of the cord contains more than its proper share of the fibres, and the anterior tract extends into the lumbar region even further than does the lateral tract. Where the direct cerebellar tract exists, this, always unaffected, limits the sclerosis of the lateral tract on the outer side. On the inner side, the "lateral limiting layer" (see p. 182) is usually much less degenerated than the pyramidal tract, and intervenes between the latter and the grey matter; but this layer suffers in some degree, perhaps by the affection of outlying pyramidal fibres contained in it. The sclerosis does not usually cease at the anterior extremity of the pyramidal tract, but extends forward in the "mixed zone" of the lateral column, lessening in intensity, and ceasing usually opposite the outer part of the anterior cornu. It is here most intense close to the grey substance, and ceases before it reaches the surface of the cord, the antero-lateral ascending tract being unaffected. This sclerosis may be due to the degeneration of the short vertical fibres that pass between adjacent regions of the anterior cornu, and which share the degeneration of the nerve-cells. Occasionally it extends, in slighter degree, into the anterior columns. The posterior columns are always free from definite degeneration, but there may be some general increase of connective tissue throughout the whole cord. The degeneration of the pyramidal tracts is usually considerable in degree; often most of their fibres seem to have perished. If it is incomplete in the cervical region, it may be slight in the lower part of the cord, the degenerated fibres being chiefly those that end in the cervical enlargement and dorsal region. When the sclerosis is intense, it is not confined to the cord; it can be traced up through the decussation (Fig. 143, B) and through the medulla oblongata, in which, as Figs. 143 and 144 show, the whole of both pyramids may be degenerated, so that they stain deeply, and no nerve-fibres can be distinguished in them. The degeneration of the pyramids has been found in several cases to extend through the pons and crus to the internal capsule, and even through the white substance to the cortex. Above the capsule, however, the pyramidal fibres are so mingled with others that their degeneration is no longer recognisable by the deeper staining of a definite tract; but it has been detected by the presence of abundant products of degeneration in the path of the fibres. In the motor cortex, the large ganglion-cells have been found fewer than normal, and distinctly degenerated, many having lost their processes, and, in the interstitial tissue, the spider cells and other connective-tissue elements are increased in number.[*] But the degeneration of the pyramidal tracts has also been found to cease at the crust[†] and at the decussation.[‡]

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[*] Koschewnikoff, 'Archives de Neurologie,' 1883, No. 18; and 'Centralbl. f. Nervenkr.,' 1885, p. 409; Marie, Neur. Centralbl.,' 1884, p. 61.

[†] Rovighi and Melotti, loc. cit.; Dornbluth, 'Neur. Centralbl.,' 1889, p. 377.

[‡] Moeli, 'Arch. f. Psych.,' x, 718.

In cases in which the symptoms of bulbar paralysis were present during life, the motor nuclei of the medulla have presented changes corresponding to those of the grey matter of the spinal cord, with a similar degeneration of the nerve-cells. In other instances, the degeneration of these nuclei has been slight, but in such cases the degeneration of the pyramids has been intense, and doubtless involved the fibres connecting these nuclei with the cortex of the brain.

The sympathetic nerves and ganglia, when examined, have been found normal, or have only presented such changes as are common apart from symptoms of disease of the nervous system.

PATHOLOGY.—The theories, according to which the disease was regarded as primarily one of the muscles or of the sympathetic nerves, have now only an historical interest. The constancy of the changes in the ganglion-cells of the spinal cord, the degeneration of the motor root-fibres, and the analogous effects of acute lesions of the anterior cornua, leave no doubt of the relation of the muscular wasting to the disease of the grey matter, and essentially to that of the ganglion-cells, and the fibres proceeding from them. The slowness of the change causes the impairment of the nutrition of the muscular fibres to proceed *pari passu* with that of the nerve-elements, and the interference with motor conduction to be proportioned to both. We are thus able to understand the condition of electrical excitability, and its slow failure, as nerve and muscle degenerate together. It is only when the slow degeneration is varied by a more acute process of destruction of cells and more rapid degeneration of fibres that the muscular tissue is for a long time less damaged than the nerve-fibres, and presents paralysis in excess of the wasting, with a voltaic irritability in excess of the faradic irritability of the nerve-endings (p. 24). Thus the essential lesion of the disease is a slow decay of the lower segment of the motor path, the segment which consists of the ganglion-cells and their prolongations in the axis-cylinders of the nerve-fibres (see p. 176). To this the conspicuous symptom, the muscular wasting, is secondary. It is perhaps better thought of as a degeneration of the whole segment than as simply a lesion of the ganglion-cells, although the latter being the element on which the nutrition of the segment depends, the two views are merely different modes of stating the same fact.*

But the disease is rarely limited to the lower segment of the motor path. The case mentioned in the note on p. 489 is an almost isolated example of such limitation—an "exception that proves the rule."†

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* In a few anomalous cases, degeneration of the motor cells of the cord has been met with when no degeneration could be detected in the peripheral mixed nerves (see Kronthal, 'Neur. Centralbl.,' 1891, p. 133). The interpretation of these cases is, at present, uncertain. Possibly, complete disappearance of some fibres left the rest apparently normal, although the skill of the investigators renders this explanation difficult to accept. Many more observations, however, are needed to justify a modification of current views.

† Moreover, the fact that these cases may not be primarily spinal must be borne in mind.

We have seen that the pyramidal tracts are commonly degenerated, and it is probable that the degeneration often extends through their entire extent, and involves the motor cells of the cortex.* Thus the upper segment is often degenerated as well as the lower segment. In the presence of that complete degeneration of the lower segment which causes the atonic atrophy of the muscles, the degeneration of the upper segment can cause no symptoms. The loss of power that it would produce is also caused by the degeneration of the lower segment, and the latter abolishes the myotatic irritability, excess of which is the characteristic indication of disease of the upper segment. Hence the degeneration of the upper segment of the motor path for the muscles that present the atonic atrophy cannot be recognised during life, for it cannot produce its characteristic symptoms.

What is the relation between the degeneration of the two segments, between the lateral sclerosis and the affection of the ganglion-cells? We cannot assume (as some have been inclined to do) that the affection of the upper segment is secondary to that of the lower, because a primary lesion of the grey matter, such as occurs in polio-myelitis, does not cause ascending degeneration of the related pyramidal fibres. Moreover, even a complete interruption of the pyramidal tracts is followed by no degeneration of their upper parts. Hence the intense degeneration met with in progressive muscular atrophy cannot be regarded as secondary. Neither, when there is atonic atrophy, can we consider the affection of the upper segment to be the primary lesion, and to be the cause of that in the lower. In the seat of slight atrophy there are, as a rule, no indications of the preceding degeneration of the upper segment, which would certainly exist if this lesion were the primary change. Moreover, degeneration of the upper segment does not necessarily cause any degeneration of the lower. It is a matter of every-day observation that intense degeneration of the termination of the upper segment may occur, from dorsal myelitis, for instance, without any considerable wasting of the legs, and such degeneration has never been known to excite the complete degeneration of the lower segment which causes atonic atrophy. Hence the only adequate explanation of the facts is that the degeneration of the upper and lower segments is simultaneous, or if not simultaneous, at least so far independent that neither is the cause or consequence of the other; both are the results of the same tendency to degeneration of the motor path. Atonic muscular atrophy is thus, at least in many cases, the visible expression of a tendency to decay of the whole motor path from the cortex of the brain to the muscles.

The weakness of some parts, as the legs, with excessive myotatic

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* At the same time, we have seen (p. 213) that the nutritional stability of nerve-fibres is less in their lower than in their upper parts, and thus their secondary degeneration may be greatest in the lower parts. That this may sometimes also be the case in these pyramidal fibres is suggested by the fact already stated that the degeneration may extend no higher than the decussation or the crura cerebri.

irritability, often going on to spasm, is explained by the degeneration of the pyramidal fibres for the legs, the lower segment being unaffected. It is easy to understand that the affection of the upper segment, and escape of the lower, or the affection of both, may vary much in relative extent, and give rise to the multiform varieties already mentioned. In such a case, the nerve-cells for the part thus paralysed are normal. This weakness of the legs usually succeeds the wasting in the arms, and in most cases in which spastic paraplegia is followed by atrophy in the arms, the latter have not shared the spastic palsy. When this condition is associated with slight wasting of the legs, without considerable change in electrical irritability, the condition exists that we have more than once considered, in which we must assume that the motor nerve cells of the cord, while structurally intact, undergo slight changes in nutrition. In this condition there are many nerve-cells of normal appearance in the grey matter; the alterations in nutrition are too slight to cause changes in aspect, or if there are such changes we have not yet learned to detect them. They are perhaps results of the degenerative changes in the termination of the upper segment; when such degeneration is secondary to a focal lesion of the cord or brain, the changes in nutrition of the cells seldom attain such a degree as to arrest the myotatic irritability, or to cause wasting such as attends the destruction of these cells.

In some cases, however, the muscular wasting may be great, although the increase of myotatic irritability persists. In these cases, as we have seen, there is considerable rigidity of the muscles throughout the whole course of their wasting, the condition that we have termed "tonic atrophy." In such a condition it is common to find that many nerve-cells have disappeared or are very small, but others remain normal or slightly changed in aspect. Apparently, in addition to the degeneration of the upper segment and to the nutritional changes just mentioned, we have then a considerable degeneration of many, but not destruction of all, the elements of the lower segment. We cannot regard this as simply secondary to the degeneration of the upper segment, for the reasons already given. It must be the expression of a distinct pathological tendency similar to that which elsewhere causes the atonic atrophy and total wasting, but slighter in extent and later in time—insufficient to prevent the less affected cells from causing rigidity under the influence of the degeneration of the upper segment. It is doubtful whether the tonic atrophy ever goes on to atonic atrophy. Theoretically conceivable, it is certain that if it ever occurs it is extremely rare. Nor does it seem that atonic atrophy ever gives place to tonic atrophy, with excessive myotatic irritability. The rigidity of tonic atrophy is due to the degeneration of the upper segment, but the effect is not produced if the lower segment is already the seat of such extensive degenerative changes as abolish myotatic irritability. The pyramidal fibres for the parts that are the seat of atonic atrophy are constantly found degenerated, although the muscles have been flaccid to the last. In the rare cases (mentioned on p. 480) in which muscles, with atonic atrophy, become rigid towards the end of the process, it is probable, as the tenderness suggests, that the rigidity is idiopathic, due to the changes in the muscles, and is not dependent on the central nervous system. It may be the result of the increase in the interstitial connective tissue, and the longitudinal division and fibrillation by which the muscular fasciculi come to resemble bundles of connective-tissue fibres. It is also possible that similar idiopathic muscular changes may ultimately, in tonic atrophy, maintain and increase the rigidity that is primarily dependent upon the spinal cord.

It has been mentioned that the cases in which the legs present the simple palsy and spasm, or the tonic atrophy, which indicate degeneration of the pyramidal tracts, have been separated by Charcot, and termed "amyotrophic lateral sclerosis," the separation being based on the assumption that in such cases the primary lesion is the degeneration of the pyramidal tracts, and that the affection of the grey matter is secondary or "deuteropathic," even where the atrophy is atonic. We have seen also that this assumption is unwarranted so far as the atonic atrophy is concerned. It is probable that the pyramidal tracts are degenerated, if not constantly, at any rate in such a very large proportion of the cases of progressive muscular atrophy, that Charcot's distinction is, in effect, giving a new name to an old disease, and that the sequence is not that indicated by the name. Whether there are indications of lateral sclerosis or not, depends on the circumstance whether the degeneration of the pyramidal fibres is or is not more extensive than the complete degeneration of the nerve-cells that causes atonic atrophy. If the latter is universal, the pyramidal tracts may be totally degenerated, and yet there may be none of the characteristic indications of such degeneration. On the other hand, both arms and legs may be the seat of the spastic paralysis that indicates pyramidal degeneration, and atonic atrophy may be limited to a few muscles of the hands. Between these we have every gradation, in degree and distribution, of atonic atrophy, spastic paralysis, and tonic wasting.

The process in the grey matter has been regarded by some as a chronic inflammation. The occasional rapid increase in the symptoms may be thought to be justification for this view; but the process in general is at the degenerative extremity of the series of nerve-lesions. The principle involved in this question has been already discussed, and we have seen that, whatever be the nature of the primary process, we must recognise secondary tissue changes of independent energy, and that a distinct process of inflammation may occasionally form part of these (see p. 396). The significance of the occurrence of inflammation may therefore easily be overrated, so far as concerns the question of the process, and the tissue-elements in which the disease begins.

DIAGNOSIS.—The simultaneous and gradual onset of weakness and wasting, the slow but progressive increase and extension of the symptoms, render the diagnosis of the developed malady simple and easy. At the onset, when only a single muscle or group of muscles is affected, the question arises whether the atrophy is local or is the commencement of a wider affection. Local atrophy is said sometimes to occur from great over-use of a muscle, especially one of the small muscles of the hand, but such a cause is extremely rare, and only to be suspected on the clearest indications.

As a rule, the local atrophy from which the affection has to be distinguished is that due to disease of the nerves. The wasting from disease of single nerves or at a plexus (as the brachial) is sufficiently distinguished by its limitation, coupled with its rapid onset and associated sensory symptoms. Much more difficult is the distinction of some forms of multiple neuritis. The difficulty presents itself chiefly in two forms:—(1) When the spinal affection begins as subacute atrophic palsy (see p. 483). (2) When neuritis affects chiefly motor branches, as in the arms in lead-poisoning, or in the legs in alcoholism, &c. In the first case it is necessary to wait for signs of slower wasting in other parts than those first affected before a diagnosis can be made. In the second, a careful search will generally reveal other symptoms of neuritis, and a known cause is usually obtrusive.

In pachymeningitis of the cervical region, with considerable damage to the nerve-roots, the wasting in the arms may resemble that of progressive muscular atrophy, and there is often weakness with rigidity in the legs, but the wasting is less chronic in onset, and is always accompanied by distinctive sensory symptoms,—by acute pains and usually by anæsthesia, irregular in distribution. The same distinctions suffice for the diagnosis in diseases of the nerve-roots of the cauda equina, as by a tumour. This may cause slow wasting in the legs, but there is always severe pain and loss of sensibility. Chronic disseminated myelitis may cause wide-spread muscular atrophy, but is distinguished by the presence of symptoms of irregular damage to other structures in the cord. The diagnosis from syringo-myelia will be considered in the account of this disease.

From primary muscular atrophy, "idiopathic atrophy," "muscular dystrophy," as it has been termed, the diagnosis is sometimes easy, sometimes very difficult. It is easy in the pseudo-hypertrophic form, and often also in the atrophic variety (the cases in which no muscles are large), on account of its characteristic distribution, its course, the age at which it begins, and the tendency to affect many members of a family, and males more than females—features that will be presently described. Whenever several cases of muscular atrophy occur in a family, or during childhood or youth, the probability is great that they are idiopathic and not spinal. Indeed, the idiopathic form is to be suspected whenever muscular atrophy begins under twenty, unless there are distinctive spinal symptoms. But cases are sometimes met with in which idiopathic atrophy begins in adult life, and the distinction of such cases may be very difficult, and will be better understood after a perusal of the account of this disease.

PROGNOSIS.—The progressive character of the malady renders the prognosis, in every case, grave and uncertain. The chief guide is the observed tendency of the morbid process, both the fact of its actual advance and the energy it manifests. At the same time, there is a possibility of arrest, greater in middle life than in old age. Some increase of atrophy in the parts already affected may occur for a short time after the process in the cord has ceased to spread—the muscular wasting going on until it corresponds to the changes in the nerves that have already taken place. The prospect of arrest seems to be greater in the cases in which the wasting is strictly symmetrical and nearly simultaneous on the two sides, than in those in which it is irregular, and attacks the second side when it has attained a marked degree in the first. Spontaneous cessation unfortunately seldom tends to occur until an advanced stage is reached; but, as the result of treatment, arrest may take place at any stage. The danger to life is chiefly proportioned to the interference with the muscles of respiration, and to the indications of implication of the medulla. Bulbar symptoms increase the gravity of the prognosis, especially when definite weakness can be recognised. Slight vague difficulty in articulation may remain stationary for years, and does not necessarily render the prognosis worse, especially if the atrophy elsewhere does not increase rapidly. If the malady ceases to advance, the prospect of any recovery depends on the rate at which the disease has progressed. Recent rapid loss of power may be to some extent recovered from, especially when the muscles present the degenerative reaction. Wasting that has existed for six months will probably persist unchanged. In a typical chronic case there is little hope of any actual recovery of tissue or power. The effects depend on a slow destruction of nerve-elements, the renewal of which seems to be impossible.

TREATMENT.—The first important element is to secure favorable conditions of life, and to maintain the general health as perfect as possible. Fresh air and gentle exercise are important, but all fatiguing exertion should be avoided, and likewise all mental strain. When the patient becomes helpless, great care is necessary. Bedsores in this disease mean inattention, and may always be avoided.

Only one method of treatment has, in my own experience, shown itself capable of arresting the disease, not indeed in all, but in more than half the cases in which it has been employed.* It is the administration of strychnia by hypodermic injection. In seven almost consecutive cases, in middle life, this treatment has been followed by arrest within a month of its commencement, and the arrest has been permanent in all the cases but one. In the senile cases the treatment has failed, but in most of them the disease was in an advanced stage, and the lumbar cord had begun to suffer. In some of the cases in which the result was prompt and distinct, strychnia given by the mouth had failed. It is conceivable that the different result is due to the fact that the agent is brought into more rapid contact with the nerve-elements, perhaps in purer form. Its action may possess greater momentum, as it were, and may thus exert an influence on the nutrition of the nerve-elements much more considerable than when it is slowly absorbed from the alimentary canal. One injection daily has been given, at any convenient place. The nitrate is the most convenient salt, one hundredth of a grain at first, quickly increased to one fortieth. It is not wise to give a larger quantity, lest the nerve-elements should be over-stimulated; although, in their damaged state, they may be unable to manifest the influence, it may yet do harm. When the malady is apparently arrested, it is well to intermit the injections for one week in three or four. Other nervine tonics seldom exert a distinct influence, but those that are useful in other degenerative diseases, as tabes, may be given by the mouth at the same time as the injections are employed. In a malady so grave, it is desirable to neglect nothing that may possibly exert a beneficial influence.

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* At first I regarded the apparent result with doubt, but careful and repeated observations have made it impossible to consider the arrest of the disease as other than the direct effect of the treatment. It may be asked, Why should not a larger dose by the mouth be equally effective? A large dose involves risk of over-stimulating the nerve-elements, and doing harm rather than good; it is quite different from a small dose acting suddenly.

Local treatment of the muscles has very little influence on the wasting, as may, indeed, be expected from its nature. The most sedulous and skilful use of electricity, voltaic or faradic, fails, as a rule, to produce alone any effect on the course of the disease. If the malady is progressing at the same rate in each arm, and the muscles of one arm are regularly treated with electricity, while those in the other arm are left alone, no difference can be detected in the rate of wasting on the two sides. It is possible, nevertheless, that electricity sometimes does a little good. In cases in which a rapid loss of power has occurred, and weakness is out of proportion to the wasting, some recovery is possible, and there is no doubt that the excitability of the muscular tissue is maintained for a longer time by galvanism, although the bulk of the muscle may not be influenced. In other cases, all that can be said is that the influence of electricity, properly applied, is in the right direction. Moreover, the disease is one of those in which patients find it hard to believe that electricity cannot help them, and the probability is that their conviction will be fostered by some medical adviser. It is one of the diseases in which unjustifiable assertions are too often made that early electrical treatment would have been successful. It may be well, therefore, if only to satisfy the patient that nothing has been left untried, that a careful course of electrical treatment should be adopted. Faradism may be used if the muscles are sensitive to it, but if they present any greater irritability to voltaism, it is better to use this. It is immaterial whether the application is confined to the muscles or whether one electrode is placed over the affected part of the spinal cord. The latter method has no disadvantages, but my own observations have failed to confirm the confident statements sometimes made regarding its superiority. It is very important that the current-strength employed should be moderate. Strong applications often cause much subsequent pain, and even increased disability, and should be carefully avoided. I have known a rapid increase of weakness follow a strong application, in a way that convinced the patient at least that the two were connected.

Rubbing and massage of the muscles have been frequently employed, and of this treatment also it may be said that its influence is in the right direction, although usually inappreciable so far as the muscular atrophy is concerned. Combined with passive movement, the influence of rubbing in preventing and diminishing deformities is more distinct. No special bath treatment is of service. When the disease occurs in the subjects of syphilis, specific treatment invariably fails, and I have even known the progress of the disease to be distinctly accelerated by an energetic course of treatment both by iodide and by mercury. It is important that all treatment should be pursued in moderation, and that its effects should be carefully watched.

Arthritic Muscular Atrophy.

Articular inflammation is almost invariably attended with rapid wasting of the muscles that move the joint. This occurs equally, whatever be the cause of the inflammation, and whether this is spontaneous or traumatic. It attends chronic as well as acute inflammations, and occurs in animals if joint-inflammation is produced in them.*

The muscles that waste are chiefly those which extend the affected joint. The atrophy is well seen in the muscles in front of the thigh, when the knee is inflamed. If the ankle is affected, the calf muscles chiefly waste; if the hip, the glutei; if the wrist, the extensor muscles of the forearm; the triceps when the elbow is affected; the deltoid when the shoulder-joint is inflamed. In rheumatoid arthritis of the finger-joints, such wasting is usually very conspicuous in the interossei, and especially in the abductor indicis. The atrophy, however, sometimes involves the flexors as well as the extensors, and rarely muscles of the limb that are near but do not move the affected joint. In very rare cases all the muscles of a limb have presented some wasting. Cases in which the atrophy is unusual in distribution, and especially when it is on the distal side of the affected joint, must be regarded with some suspicion, because inflammation may have spread from the joint to a nerve, and distant wasting may have been thus produced. It is said that there is sometimes, at the onset, a considerable weakening of the muscle, interfering with movement more than can be accounted for by the pain in the joint which movement causes, and that such initial palsy is transient (Valtat, Duchenne). But the pain, by its inhibitory influence, usually obscures such palsy, which is indeed confessedly rare.

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* Valtat, 'Archives Générales,' 1877, tome xxx, pp. 159 and 321. The subject has been discussed by Vulpian ('Leçons sur l'App. Vaso-moteur,' 1875, t. ii) and by Paget ('Lancet,' 1873, vol. ii, p. 727, in a lecture republished in 'Clinical Lectures and Essays,' 1875, p. 208).

The atrophy, on the other hand, may be regarded as almost constant. If the onset of the joint affection is acute, the wasting occurs rapidly; in a week or ten days, a difference in the circumference of the limb may be detected by measurement. However long the affected muscle may be, the wasting involves the whole length of it, and not merely the part in the neighbourhood of the diseased joint. The degree varies; usually moderate, and sometimes slight, it is occasionally considerable, so that the femur may be readily felt when the quadriceps femoris is the seat of the wasting, or the head of the humerus may be distinct, and the acromion prominent, if the deltoid is affected. Although the whole length of the affected muscle is always involved, if the muscle is a wide and compound one, some parts may suffer chiefly. Thus in the thigh all parts of the extensor may waste equally, or the rectus or vastus internus may suffer more than the other parts. The wasting increases during two or three weeks, then becomes stationary, but continues as long as the joint disease lasts. When the joint has recovered, the muscles in most cases slowly regain their normal size. Occasionally, especially when the arthritis has lasted a long time, the wasting may continue for months or years after the joint disease is at an end.

The electrical irritability of the atrophied muscles may be normal, but is often slightly lowered, equally to faradism and voltaism. The change is trifling, to be recognised only by comparison with the other side. There is generally a distinct, and sometimes a considerable, local increase in myotatic irritability; the knee-jerk is excessive if the thigh muscles are affected, and a rectus-clonus can sometimes be obtained. A foot-clonus may be elicited when the ankle-joint is affected. Occasionally the increased irritability extends beyond the region of atrophy, so that, for instance, a foot-clonus may be obtained when the knee-joint and thigh muscles are affected, although there is no wasting below the knee. As an example of this, which illustrates also the occasional persistence of the atrophy, may be mentioned the case of a young man who jumped over a hoarding and twisted his left leg in doing so. He felt immediately severe pain in the knee, the joint quickly swelled, and became the seat of an acute inflammation which lasted for several weeks, and then slowly subsided. During the inflammation, the thigh wasted. I saw him two years after the onset, and there was still considerable wasting, involving the whole of the extensor of the knee. The minimum circumference of the left thigh above the knee was three quarters of an inch less than that of the right, although there was no difference between the two legs below the knee. There was a slight diminution in faradic and voltaic irritability in the affected muscles. The knee-jerk was much more considerable on the left than on the right side, and there was a well-marked foot-clonus in the left leg, but none in the right. A year and a half later the symptoms were unchanged. Very rarely some contracture occurs in the opponents of the atrophied muscles. Sensory symptoms are as a rule absent, but there may be slight tingling in the skin during the acute stage; it is said that areas of anæsthesia sometimes develop, but probably in such cases, inflammation has extended to a nerve in the vicinity of the joint.

PATHOLOGY.—Few facts have been ascertained regarding the condition in man. In one case, carefully investigated, the only change was in the muscles, and consisted merely in a narrowing of the fibres, one half of which were below the normal average, while no less than 84 per cent. fell short of a standard which, in health, only 21 per cent. failed to reach.[*] A few presented longitudinal striation (as in Fig. 140), and some of normal width were unduly tortuous; otherwise their aspect was normal. The sheath nuclei were proliferated in places, and, apart from this change, the intermediate substance presented local increase in quantity, especially where the fibres were narrower than normal. This observation agrees with those that have been made on animals, in which the condition has been produced, and in them, moreover, the motor nerves have been found normal (Valtat).[†] Indications of inflammation have been found in the joint-nerves,[‡] as might, indeed, be expected, whatever the mechanism of the atrophy. Visible changes in the spinal cord have never been discovered, but this does not exclude nutritional changes.

We may, therefore, exclude from the possible causes all forms of motor neuritis, both simultaneous, due to the cause of the joint inflammation, and consecutive, communicated to the adjacent nerve from the joint. The latter (a secondary extension of inflammation to the nerve) would manifestly not explain the affection of the whole of a muscle on the proximal side of the affected joint—as, for instance, atrophy of the whole extensor in arthritis of the knee,—although the extension does occur and must be recognised as an occasional event.[§] We must also recognise the possibility of a simultaneous neuritis, with acute changes in the nerve-fibres, manifested by the reaction of degeneration in the muscles. The event is, however, exceptional. It is certainly separable and to be separated from the common arthritic atrophy; but it is not easy to keep apart the two varieties of neuritis—the simultaneous and the consecutive—when a case is seen only long after the onset, because a neuritis that arises by extension may travel along the nerve and present an ultimate distribution quite different from that which it possessed at the onset.

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[*] Darkschewitsch, 'Neur. Cent.,' 1891, p. 353.

[†] Valtat, 'De l'Atrophie Musc., &c.,' Paris, 1877. See also Deroche, 'Etude clin. et exp.,' Paris, 1890.

[‡] See Duplay and Cazin, 'Arch. Gén.,' January, 1891.

[§] *E.g.* to the ulnar, in rheumatoid arthritis of the hand (Bury, 'Med. Chron.,' 1888, p. 182). See on this subject A. E. Garrod, 'Med.-Chir. Trans.,' lxxi, 265; also Pitres and Vaillard, 'Rev. de Méd.,' 1887, No. 6.

For ordinary arthritic atrophies we must seek some other explanation. The nature of their cause long ago suggested to Paget a "reflex influence" on the muscles as the probable mechanism, and Vulpian and Charcot have suggested a similar hypothesis, assuming a derangement of the nutrition and influence of the motor cells of the cord, to be determined by the morbid impulses from the joint-nerves, and to determine the alterations in the muscles. The theory receives important support from the fact that the wasting of the muscles is prevented by previous division of the posterior spinal roots (Raymond), which amounts, indeed, to an indirect demonstration of the fact that a reflex process* underlies the phenomena. It is doubtful whether we can go much further than this. By preceding hemisection of the cord, it is increased on the side of the operation; but the conditions are here more complex, and the demonstration of the reflex relation less simple, than in the result of division of the roots.

The increased knee-jerk, &c., that attend the wasting, and the curious fact that arthritis may set up a degeneration of the spinal cord, apparently beginning in the termination of the pyramidal fibres (see p. 443), should be kept in mind. They are certainly significant, and suggest that the influence from the joint-nerves acts on the "controlling structure" of the muscle-reflex centre (see p. 201), but more facts are needed before a valid hypothesis can be framed.

DIAGNOSIS.—The moderate degree of the wasting, with corresponding change in electrical reactions, coupled with the wide extent of the alteration, embracing the whole of the muscles involved, constitute a distinctive characteristic—distinctive, at least, when taken in connection with the preceding joint affection which is the cause of the atrophy. A primary neuritis, causing secondary arthritic adhesions, has its own features—their wider range, so far as function is concerned, and especially their limitation to nerve distribution. Hardly any other malady is likely to be confused with it, except some graver disease, while still in an early stage, but such an affection can only be distinguished by waiting until the nature of the malady has had time to show itself.

PROGNOSIS.—When the inflammation of the joint is brief in duration, recovery of the muscles may be anticipated with confidence. In children, even after prolonged joint disease, the muscles usually regain their normal bulk. In adults the wasting often lasts for a long time after the joint is well, and a cautious prognosis should be given if the arthritis has lasted long. Even if slight wasting is persistent, normal power is usually recovered, but, as the case mentioned above shows, the symptoms sometimes continue for years. The prognosis should be especially cautious where there is a marked increase in myotatic irritability, or if the atrophy shows a disposition to involve other muscles of the limb than those concerned in moving the affected joint.

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* Raymond, 'Rev. de Méd.,' 1890, 374.

TREATMENT.—The chief treatment is local; electrical stimulation of the muscles, and gentle rubbing. It is doubtful whether drugs have any influence on the condition, but small doses of strychnia may be given, or added to whatever agent is given for the joint affection. As long as the joint is inflamed, treatment rarely causes any increase in the bulk of the muscle. It is of great importance to secure the early recovery of the joint, and to avoid all influences that cause pain in it, since, as we have seen, it is apparently through the sensory nerves that the joint affection exerts its injurious influence. When the arthritis has ended, the muscles usually recover slowly without assistance, but it is probable that the local treatment accelerates the process. The form of electricity is of little consequence; either faradism or voltaism may be employed, but should only be used in sufficient strength to cause gentle contraction. The muscles act readily to faradism, and a mild faradic current is, as a rule, the best to employ. All painful stimulation of the sensory nerves should be avoided.

MUSCULAR ATROPHY FROM OVER-USE.

Muscles that are much used sometimes waste. The effect is seen most frequently in the small muscles of the hand, especially in persons of weakly constitution, who use these muscles unduly. Thus a young lady devoted many hours a day to "illuminating," during several years, and then some muscles of the thenar eminence began to waste, and became considerably atrophied. Such wasting is scarcely ever met with in the larger muscles, but has been observed in the biceps, consecutive to hypertrophy, in Sheffield smiths (Frank-Smith). The electric irritability is gradually lowered in this local wasting, equally to both currents, as it is in progressive muscular atrophy. We do not know whether this wasting from over-use is purely local, or whether the related ganglion-cells of the spinal cord fail first, and the muscular wasting is secondary to their atrophy. As Gull said many years ago, "it is as reasonable to infer a lesion of the grey matter from overwork as of the muscles."* The wasting often persists when the over-use of the muscles is discontinued. Hypertrophy from over-use does not usually give place to atrophy. These two facts are somewhat in favour of the view that the lesion is primarily of the nerve-cells. The wasting in these cases shows no tendency to extension beyond its original limits. The treatment is, first, the cessation from the excessive exertion of the muscles; secondly, the improvement of the general health, and the administration of nerve tonics, especially of strychnine; and thirdly, the gentle electrical stimulation of the muscles by either faradism or voltaism.

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* 'Guy's Hosp. Reports,' 1862, p. 246.

IDIOPATHIC MUSCULAR ATROPHY

(MYOPATHIC ATROPHY; MUSCULAR DYSTROPHY).

Idiopathic atrophy, which is primarily muscular, although it is not one of the diseases of the nervous system, is commonly described with them, because it frequently presents so close a resemblance to the spinal atrophy just described as to be distinguished with some difficulty. Moreover, even the varieties that do not present this close resemblance were for a time, and indeed until lately, believed to be of central nature, to depend on the spinal cord, and to be rightly included among its diseases. Hence the custom of thus describing them has become established, and has now to be followed, although we know that it is based on a mistaken theory. At the same time, not only has the method the advantage of convenience, but it has also some measure of scientific justification. The muscles are more closely allied to the nerves than to any other structures, as regards both physiological properties and pathological susceptibility, and it is doubtful whether any other association would better befit these diseases.

The idiopathic myopathies have received a great amount of attention during the last seven years, and more facts have been ascertained than can yet be used. The difference of opinion regarding the relation of the several forms is still considerable. For the present, it seems better, therefore, to leave unchanged the method of describing them which was first adopted, and which is not without its advantages. Before long, it may be possible to recast the description of these diseases on lines of greater accuracy.* The following account of them may, however, be more intelligible to the reader if it is preceded by a brief sketch of their general pathology. Any points of interest in connection with their history, &c., are left to be mentioned when the varieties are described.

All the diseases of this class seem to depend on a defective tendency of development of the germinal tissue which forms muscles; they are

* A treatise upon them by Erb, who made a special study of the subject, is now in course of publication, and cannot fail to make our knowledge far more precise

essentially congenital diseases. Although they are, in most cases, merely potential maladies at the time of birth, and sometimes for years afterwards—even occasionally, during a considerable part of life—the morbid tendency does sometimes attain actual development in the earlier years. But we know nothing, or almost nothing, at present, of any other cause than this developmental tendency; we know nothing of these diseases as acquired maladies, the result of influences acting on a healthy and normal organism.

Another fact of their general pathology is analogous to that which we have already considered in connection with developmental diseases of the central nervous system. It depends on the double constitution of muscular as of nervous organs, on the presence of interstitial connective tissue between the contractile elements, and on the relation of both to trophic tendencies. An overgrowth of the interstitial tissue may concur with wasting of the fibres, and this overgrowth may be abnormal in its features. Hence Erb has chosen the term "muscular dystrophy," as a more precise designation for the class; we have yet to see whether it will obtain general acceptance. The important fact to recognise is that there may be either a simple defect in the growth of the fibres, ultimately leading to their disappearance, or there may be, with this, an overgrowth of the connective tissue, either a simple hyperplasia, or a perverted growth in which fat-cells form. These cells increase its bulk, but are ultimately removed. The muscular fibres are thought sometimes to present hypertrophic enlargement, but simple wasting is the most common change.

The symptoms by which these changes are manifested vary according to the nature of the alteration. In all cases, muscular power fails as the contractile fibres waste. The chief difference in aspect, however, depends on the seat of the affection and the effect of the interstitial changes, and especially on the frequent enlargement of muscles from the formation of fat in the fibrous tissue. This produces the semblance of hypertrophy that has led to the name "pseudo-hypertrophic paralysis" being applied to the cases in which it is present. The amount of enlargement varies much; the fibrous tissue alone may cause none, but it is sometimes great and wide-spread in the cases in which fat is formed. In all, however, a primary shrinking of the muscles is due to the wasting of their proper fibres, the result mainly of a defective tendency of vital endurance or growth, and only in a slight degree to the influence of the interstitial tissue, when this is increased in quantity.

Thus certain varieties are constituted by the external manifestation of the muscular changes. Even in the class in which pseudo-hypertrophy occurs, there may be no change, or only diminution in the size of the muscles, but with increased firmness, as the accompaniment of the lessened power. When there is enlargement, this may or may not correspond in time to the loss of power, since the muscular fibres may waste either during interstitial growth, or after this tissue has undergone the atrophy which ultimately supervenes. Thus we may have two classes of cases belonging to this type, one with enlargement of muscles, few or many, the other with only wasting of muscles, the two corresponding in position and in the general course and relations of the affection.

In another form there is never an interstitial growth of fat, and, probably, no considerable increase of fibrous tissue; simple atrophy of the fibres is its pathological characteristic, while primary shrinking of the muscles is its external manifestation. The shoulder muscles are prone to suffer most, and in some cases there is a peculiar affection of the face. These cases are also more variable in the time of life at which the symptoms begin. But these varieties, as we shall see, are connected by cases which to some extent combine the various features. Such combined forms forbid the separation of the types, which, nevertheless, frequently maintain so distinct a course in many members of a family, even through several generations, that we are compelled to recognise their distinctness, although we cannot divide them altogether.

The essential element in the disease has been spoken of as a "qualitative" defect, which entails an imperfect development, manifested sooner or later by the defective vitality of the proper elements of the tissue. But there is reason to believe that the defect is not always merely qualitative. In some cases the defect in certain muscles is so absolute at so early a period, as to make it almost certain that these muscles or parts of muscles are congenitally absent, and that the germinal defect is thus quantitative as well as qualitative. The muscles in which this apparent failure is observed vary in the several forms, and will be mentioned in the account of these.

The peculiar form of atrophy, which differs from the others in the early affection of the muscles supplied by the peroneal nerve, is separately described, and its features are not included in these remarks.

PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS (LIPOMATOUS MUSCULAR ATROPHY, &c.).

The affection thus designated usually manifests itself during the later developmental period of childhood, and clearly depends on a morbid developmental tendency, which is often present in several members of the same family. It is characterised by a progressive change in the size, and diminution in the power, of many muscles. The apparent cause of the change has been already mentioned.

Isolated cases, which can now be recognised as examples of this disease, were recorded in England in 1830 (Sir Charles Bell) and in 1847 (Partridge), and in Italy in 1838. A series of cases was described by Meryon in 1852, and Oppenheim in 1855, but enlargement of the muscles was not conspicuous in these.[*] Several remarkable examples had already come under the notice of Duchenne, who was busy exploring the field of muscular paralysis with the aid of "localised electrisation." He recognised its novel features, and published an account of it in 1861, under the name by which it has since been generally known.

Etiology.—Our knowledge of the causes of the disease is limited to a few general facts. Males furnish the majority of the cases; they suffer at least four, and perhaps seven times as frequently as females. In the latter, moreover, the malady is slighter in degree, later in development, and less frequently causes death.

The disease occurs rather less commonly in isolated cases than in family groups. The number in a family has varied from two to eight. As many as eight brothers suffered and died in the family described by Meryon, while all the daughters escaped. In a family known to me, four sons have suffered and none of the daughters; in another instance two daughters are affected and no sons. Thus there may be a tendency in a family to the affection of one sex, and not the other; but, on the other hand, children of both sexes may suffer in the same family. In many instances in which several members of one generation are affected, no antecedent cases can be traced in the family; the malady, while congenital, is not hereditary. In other families antecedent cases can be traced, and these are invariably on the mother's side. The disease is thus transmitted by women who are not themselves its subjects. In a case in which four brothers suffered, the mother's brother and sister were likewise affected. Again, a brother and sister were diseased, one daughter of a second sister, and three daughters of a third sister. In another instance, a boy suffered, and his sister, unaffected, had two sons diseased and a daughter free, of whose children two sons were the subjects of the malady. Thus the congenital tendency is exclusively due to the maternal element in the embryo. This is also shown by a fact many times observed, that the children of the same women, by different husbands, suffer in the same way.

Indirect hereditary tendencies, such as are indicated by the occurrence of diseases of the nervous system, can be traced so rarely that it is doubtful if they have any influence. Neither the age of parents, nor their intemperance, appears influential, and their consanguinity becomes effective only when raised in energy by repetition. Thus in a family known to me, the intermarriages during five generations were very numerous, and of eight children in the present family, five are albinoes, and two of these are the subjects of pseudo-hypertrophic paralysis.

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[*] Nevertheless, Meryon's cases ('Med.-Chir. Trans.,' 1852) certainly belonged to this variety. Conclusive proof of the fact is afforded by cases in collaterals which have come under the writer's observation in near and distant branches of the same family. Other evidence of the fact is described in a 'Clinical Lecture on Pseudo-hypertrophic Paralysis' (London, Churchill, 1879). The chief literature is referred to in that lecture; the numerous papers that have appeared since have added chiefly to our knowledge of the characters of the allied forms of idiopathic atrophy, &c., and the relations of these to the pseudo-hypertrophic variety, and are referred to on a later page.

The disease always manifests itself during the period of development, sometimes in the early stage of growth, at the close of infancy, often only during mid-childhood, rarely not until growth is nearly ended. In a third of the cases, the first symptoms are noted when the child first attempts to walk, which is usually a little later than in healthy children; very rarely indeed, the child has never walked. In about another third the child seems well until it is four, five, or six years old, and then impairment of power attracts attention. In three quarters of the cases, the disease manifests itself before the tenth year. Rarely, the patient is conscious of no symptoms until after puberty, at the age of eighteen or twenty, but in such apparently late onset, there has been enlargement of muscles long before power becomes impaired, and the disease began much earlier than it seemed to do. One patient, for instance, in whom weakness was only noticed when she was twenty, had been often "chaffed," when a young girl at school, on account of her "tea-kettle calves." Neither social state nor general constitutional condition seems to influence the occurrence of the disease, but its manifestation has sometimes been apparently accelerated by influences that disturb the general health; it has been first noticed, for instance, during convalescence from some general illness.

SYMPTOMS.—Impairment of power usually attracts attention before any change is observed in the size of the muscles, or if these are noticed to be large, it is with feelings of parental pride rather than with suspicion, in spite of the fact that the children often walk clumsily, fall with ease, and rise with difficulty. The act of going upstairs is especially difficult to them; the child has to take hold of the banisters and pull himself up.

The muscles may at first present nothing unusual, especially in slight cases, or if the child is fat, as is frequently the case. But at the age of five or six years an unnatural enlargement of certain muscles is usually conspicuous, especially when there is a contrast between these and other muscles which are small. If enlargement is almost universal, it is usually great and conspicuous. The enlarged muscles usually for a time become still larger in comparison with the others, but afterwards they cease to increase and ultimately become smaller, first relatively and then absolutely. This change occurs earlier in some muscles than in others, and it may give place to a condition of distinct atrophy.

Among muscles that are most frequently large, those of the calf take the first place. They sometimes attain a remarkable size. I have measured a calf 14½ inches in circumference in a boy of twelve. The muscles in front of the lower leg are less frequently enlarged, but sometimes project beyond the edge of the tibia. The extensors of the

FIG. 145.

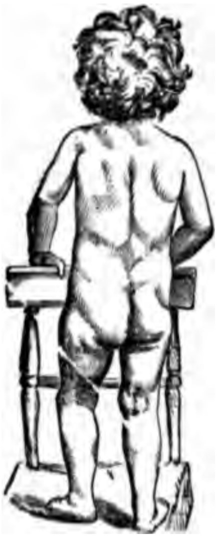


FIG. 146.

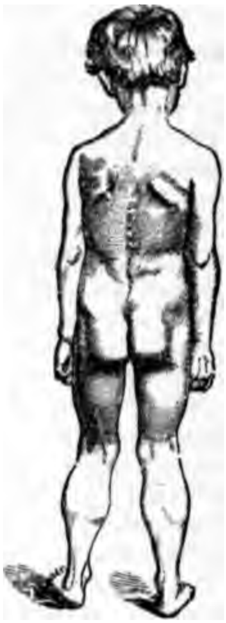
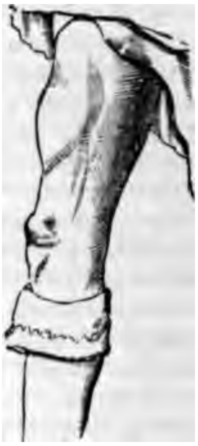


FIG. 147.



FIGS. 145 and 146.—Two brothers, aged four and seven, suffering from pseudo-hypertrophic paralysis.

FIG. 147.—Partial enlargement of rectus, the vasti being small.

knee are often big; occasionally the rectus or vastus internus is alone increased in size (the rectus in Fig. 147), and the other parts may be normal or small; less frequently all parts are small. The flexors of the knee commonly escape. The glutei are frequently conspicuously large; the flexors of the hip are, of course, inaccessible to observation, but they are usually feeble, and no doubt diseased; there is generally enlargement of the lumbar muscles, and disease of a peculiar and important character in those of the shoulder.

Of all the muscles of the body, next to those of the calf, no one is enlarged more frequently or in greater relative degree than the infraspinatus. It often stands out so conspicuously that its edge is apt to be mistaken for that of the scapula (Figs. 148 and 149). The supraspinatus is sometimes also prominent, but its condition is usually concealed by the trapezius, which is little involved. The deltoid is also frequently large; the serratus rarely. The pectoralis is never enlarged, but, on the other hand, its lower half is wasted or absent (see p. 518) in a large proportion of the cases, and with this the latissimus dorsi, which has the same action in depressing the raised arm (see p. 26). The teres major may share the wasting of the latissimus.

The other muscles of the arm suffer in diminishing degree and frequency from above downwards. The triceps and biceps are sometimes enlarged, the former more frequently than the latter, but occasionally only in one part. Both these muscles are sometimes wasted. The forearm muscles suffer in only a small minority of the cases, and the intrinsic muscles of the hand usually escape altogether.

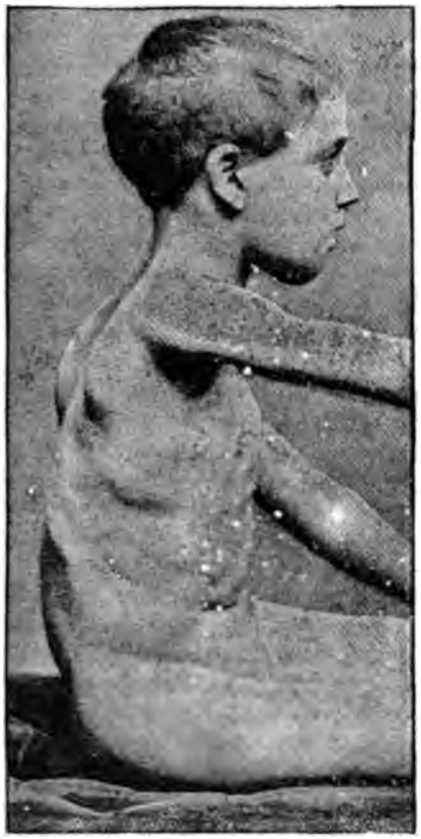


FIG. 148.— Pseudo-hypertrophic paralysis. Absence of latissimus dorsi, enlargement of infraspinatus. (From a photograph.)

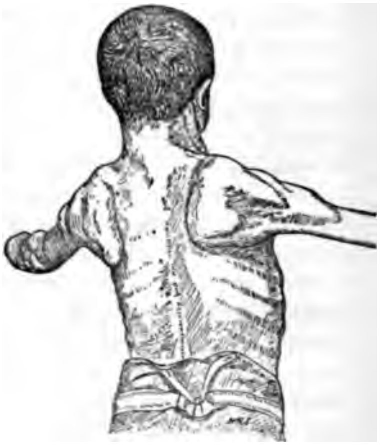


FIG. 149.—Wasting of latissimus dorsi and serratus; enlargement of infraspinatus, supraspinatus, and deltoid; atrophy of biceps and triceps. (By Dr. H. R. Spencer, from a photograph.)

This escape of the intrinsic muscles of the hand affords a very marked contrast to spinal muscular atrophy, in which they suffer early; but no rule is free from exception, and although the escape of the hands in the idiopathic affections is almost constant, it is not quite invariable; slight (or commencing) implication of the intrinsic muscles has been met with in very rare instances that were otherwise typical,* and quite distinct from the "peroneal type" to be presently mentioned.

The muscles of the neck are very seldom affected, but I have noted, in a few cases, wasting of the clavicular part of the sterno-mastoid. Those of the face do not suffer except in extremely rare cases intermediate between this and the third type (see also Westphal, 'Charité Annalen,' 1887, xii, p. 447), but in the patient shown in Fig. 152 there was considerable enlargement of the masseters. The tongue has been increased in size in a few instances. The other muscles supplied by the cranial nerves always escape.

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* *E.g.* Sachs, 'New York Neurol. Soc.,' Oct. 2nd, 1888; Baumler, 'Sudwest. Neurol. Versamml.,' Freiburg, 1888. I have once met with wasting of the extensors of both phalanges of one thumb, and enlargement of the abd. indicis has been observed (Taylor, Clin. Soc., April 24th, 1891), and fatty growth in the thenar muscles (Berger, 'Arch. f. Psych.,' Bd. xiv).

The diseased muscles are weak, but the impairment of power is to some extent irrespective of the change in size. The muscles that are abnormally small are generally weaker than those that are abnormally large; and in the latter the weakness increases with the wasting. In the legs, the greatest weakness is in muscles that are inaccessible to examination—the flexors of the hips; next in order of weakness come the extensors of the knee, and the extensors of the hip. The muscles below the knee usually retain considerable power for a long time, and the extensors of the ankle fail before the flexors. In the upper limbs the depressors of the arm are usually alone weakened during the early period of the disease, but subsequently the shoulder muscles suffer, then the triceps and the biceps, while the muscles that move the hand commonly retain good power to the last.

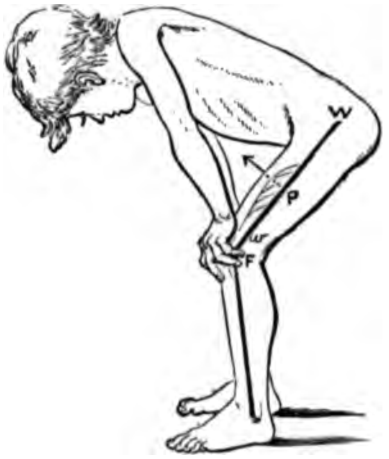

FIG. 150.—Mode of obtaining extension of hips in pseudo-hypertrophic paralysis. F, fulcrum of the lever formed by the femur. P, mean position at which the power is applied by contraction of the quadriceps femoris. W, position of weight in the ordinary mode of rising. w, the place to which part of the weight is transferred by putting hands on knees.

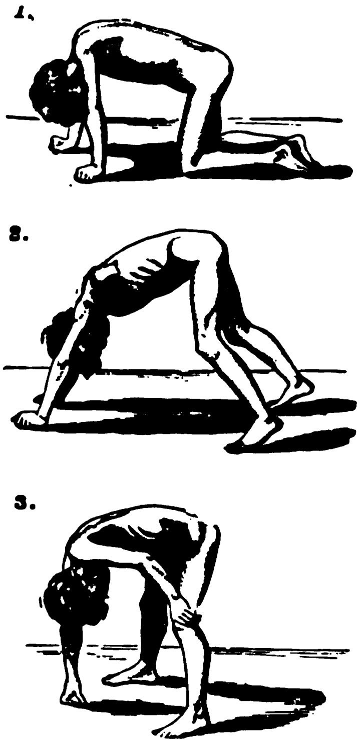

FIG. 151.—Mode of rising from the ground in pseudo-hypertrophic paralysis.

The distribution of weakness in the legs causes certain peculiar defects of movement which are very characteristic, and some are even all but pathognomonic of the disease. The difficulty in going upstairs is especially due to the weakness of the extensors of the knee and hip. The defect of the extensors of the hip causes the gait to have a peculiar oscillating character, in which the body is so inclined as to bring the centre of gravity over each foot, on which the patient successively throws his weight, because the weak gluteus medius cannot counteract the inclination towards the leg that is off the ground, unless the balance is exact. The greatest defect, however, is in the power of rising from the floor, and the most characteristic peculiarity is the mode in which this is achieved, if it be still possible, and no objects are near, by which the patient can aid himself. He commonly has not sufficient power to extend the knees when the weight of the trunk is on the upper extremity of the femur, which is then a lever in which the power, applied between the fulcrum and the weight, acts at least advantage. He therefore places his hands on his knees, as in Fig. 150; and his arms thus bring much of the weight of the upper part of the trunk on the femur close to the fulcrum, between this and the power, which can then act at greater advantage. Moreover, the mere weight of the head, which is in front of the arms, tends to aid the extension. This, indeed, may effect the extension of the knee without the aid of the extensor muscles, as anyone may ascertain by observing the mobility of the patella in this attitude. When the knees are extended, the power of the extensors of the hip may be sufficient to raise the body into the upright position, or the patient may aid them by an upward push with the hand as he takes it off. If, however, these extensors are weak, the hands are often moved higher and higher up the thighs, grasping alternately, and thus pushing up the trunk. To get thus the requisite support, the knees must not be quite extended; and if their extensors have no power, the device cannot be employed, and the patient is altogether unable to rise. In many cases, especially when extension of the hip is easy, the patient achieves the extension of the knees in another way; he puts the hands on the ground, stretches out the legs behind him far apart, and then, the chief weight of the trunk resting on the hands, by keeping the toes on the ground and pushing the body backwards, he manages to get the knees extended, until the trunk is supported by the hands and feet, all placed as widely apart as possible (Fig. 151, 2). Next the hands are moved alternately along the ground backwards, so as to bring a larger portion of the weight of the trunk over the legs. Then one hand is placed upon the knee (Fig. 151, 3), and a push with this, and with the other hand on the ground, is sufficient to enable the extensors of the hip to bring the trunk into the upright position.

The shortening and contraction of certain muscles lead to another group of symptoms,—distortions due to permanent alteration in the position of joints. Some of these are produced, as are distortions in other forms of muscular weakness, by shortening of the less affected opponents of the weaker muscles. Thus the knee-joints become fixed by the contraction of the flexors, and the elbow by the contraction of the biceps when the triceps has lost all power. These contractions only occur late, and are usually facilitated by the habitual flexion of the knee- and elbow-joints. But the deformity at the ankle-joint, which results from contraction of the calf muscles, commences earlier, before their opponents are weak, and is the result of shortening of the muscles. As a consequence of it, the patient cannot get the heels well upon the ground, and the foot cannot be flexed passively beyond a right angle. The gradual increase of the contraction results in considerable "talipes equinus," and as power lessens, the patient is able to walk less, and the consequent loss of the extension involved in the act permits a rapid increase in the contraction. The feet, as Fig. 152 shows, soon assume a posture of extreme extension, the dorsum being in a line with the front of the leg, or forming with it a convex curve. A subluxation of the ankle-joint takes place, and the articular surface of the astragalus, its anterior extremity, and that of the os calcis, form three prominences under the skin. When this reversal of the ankle occurs, the tibialis anticus can no longer act as a flexor.

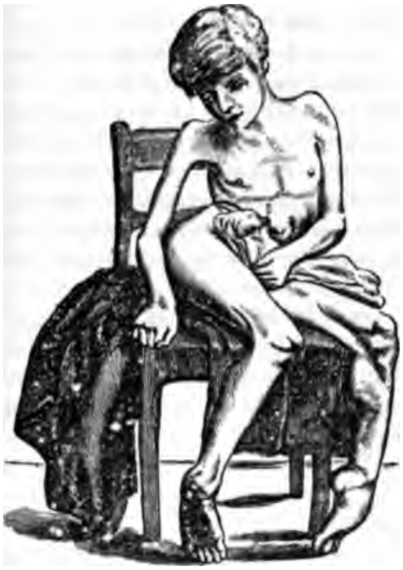

FIG. 152.—Late stage of pseudo-hypertrophic paralysis; a boy fourteen years old, with muscular contraction and wasting, and lateral curvature of the spine.

Another deformity, which is due chiefly to muscular weakness, is curvature of the spine. An antero-posterior curve, with the concavity backwards, is an early symptom of the disease, and it may become extreme, the upper part of the trunk being carried so far back that a vertical line from the scapula falls an inch or more behind the sacrum. It is due, not to the weakness of the trunk muscles, but to that of the extensors of the hip, in consequence of which the pelvis is inclined forwards, carrying with it the lower lumbar vertebræ; hence the upper part of the trunk has to be held far back to keep the centre of gravity of the body over the feet. The proof of this mechanism is that when the patient sits, and the pelvis is supported on the ischial tuberosities, the lordosis disappears. It is, indeed, replaced by an opposite curve, in which the back becomes convex, clearly due to the weakness of its extensor muscles. This curve may become very great, as in the case shown in Fig. 153. The weakness of the spinal muscles also permits the occurrence of lateral curvature (Fig. 152), influenced, in its direction, by the habitual posture and the preponderance of weakness on one side or the other.

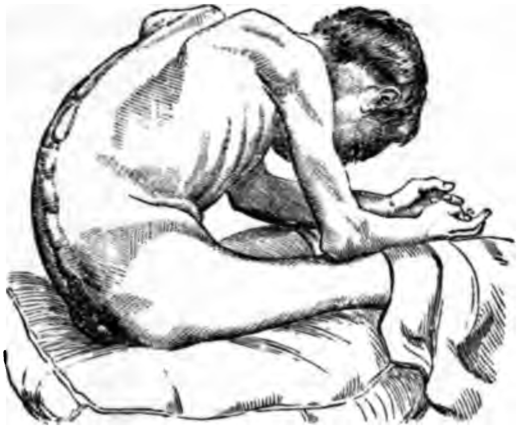

FIG. 153.—Lad, aged fifteen; late stage; wasting of thighs; inability to sit upright in consequence of the weakness of the spinal muscles.

The electric irritability of the muscles is only altered when distinct weakness or wasting has set in, when it is lowered alike to faradism and voltaism. There is never any trace of degenerative reaction.

The knee-jerk may be at first normal, but as the extensors of the knee become feeble, it is always lessened and gradually disappears. It is never excessive, and in all advanced cases it is lost. Sensation is unaffected, and so also are the sphincters in the vast majority of cases. Very rarely there has been, towards the end, a slight difficulty in the retention or expulsion of urine, to be regarded, perhaps, rather as a complication than as an effect of the disease. All other functions of the nervous system are commonly normal, including those of the sympathetic. The mental development of the subjects of this disease is generally beyond that of other children of the same age, doubtless on account of the indirect influence of a malady which withdraws them from active amusements; mental defect is a pure complication.

The rate of progress of the disease and its duration vary much. After some years, often between ten and fourteen, the power of standing becomes lost in consequence of the increasing weakness and the contraction of the calf-muscles. When the patient ceases to walk, the muscular disease makes more rapid progress, deformities become greater, and the patient may become almost helpless, except in the hands, and yet live on for several years. Death is sometimes due to some intercurrent malady, as an acute specific disease, but generally the lessened respiratory power causes some chest affection to develop, or one that should be trifling, to become grave. Life is thus ended by acute pneumonia or bronchitis, or by chronic lung disease,—a form of pneumonic phthisis or broncho-pneumonia, which develops gradually, with little febrile disturbance. There is never sufficient paralysis of the respiratory muscles to cause death directly. The duration of the stage of helplessness depends very much on the care which the patient can obtain.

In the cases in which muscular power remains good until after puberty, the progress of the disease is generally slow. The patient may reach the age of thirty before power is much impaired. It is possible that, in some cases, the disease never attains a considerable degree. More frequently, however, after slight symptoms have lasted for some years, a rapid increase occurs, and very few patients reach the age of forty. The course of the disease is slower in girls than in boys, and females furnish a relatively large proportion of the late cases.

Varieties.—The chief varieties of the disease depend on the age at which it commences, and on the condition of the muscles, whether they are large or small. In rare cases a single muscle may be large, and the rest small, as in Fig. 154, in which only the vasti were increased in size. Or everywhere and from the first the muscles are smaller than normal, and they progressively waste. Such cases are not uncommon; in the first group of cases described (by Meryon), enlargement of muscles was inconspicuous. Many cases in which all muscles are small belong properly to the form considered in the next section. The cases described by Meryon must be regarded as examples of the pseudo-hypertrophic form (see p. 505), but they present many points of resemblance to the "simple atrophy" described further on.

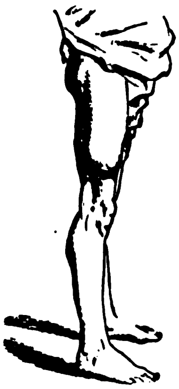

FIG. 154.—Enlargement of the vasti and not of the rectus. In this case all other muscles were below normal size.

Complications.—Congenital mental weakness, due apparently to defective development of the brain, sometimes complicates pseudo-hypertrophic paralysis. In rare cases there have been indications of some other morbid condition of the central nervous system, such as epilepsy. It is uncertain in what light the slight occasional affection of the bladder is to be regarded, whether as an invasion of the vesical muscles or as a central complication. Vigoroux has recorded a case in which the symptoms of pseudo-hypertrophic paralysis were combined with the peculiar rigidity of Thomsen's disease. Of course the subjects of the disease are liable, like other children, to various affections of the nervous system; I have seen both chorea and polio-myelitis as merely accidental complications.

PATHOLOGICAL ANATOMY.—It is rare, at the time of death, for any muscles to be actually larger than natural, and most of those that are affected are usually below the normal size. They are pale and yellowish in colour, and often, to the naked eye, resemble perfectly masses of adipose tissue. The resemblance is not merely one of aspect. As seen under the microscope, it may be difficult for the observer to realise that he is not looking at a fatty tumour. Nothing may be at first visible but fat-cells, precisely like those of adipose tissue. Among the cells, however, are tracts of nucleated fibrous tissue, and a closer examination of these shows that the tracts contain also muscular fibres (Fig. 155), most of them much narrower than normal. They are also irregular in width; a broad fibre, for instance (as in the figure), suddenly becoming narrow.

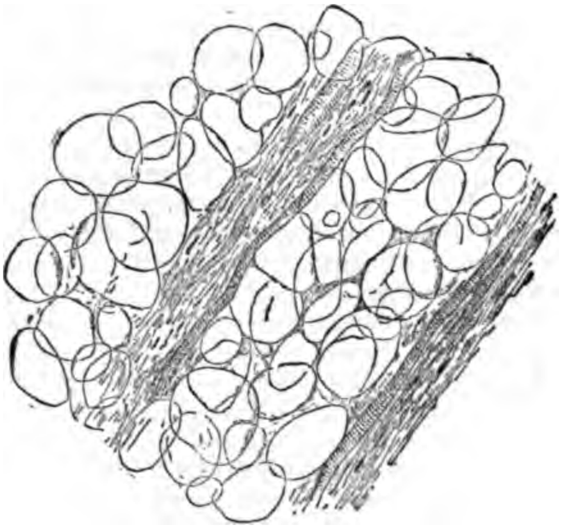

FIG. 155.—Gastrocnemius muscle; muscular fibres, irregularly narrowed and in part degenerated, lie among tracts of nucleated fibrous tissue, separated by adipose tissue.

The fibres for the most part preserve their transverse striation, but where they are narrowest this may have in part disappeared, either by granular degeneration, or, more commonly, by a simple fading of the striæ. In the narrowed fibres the striæ are sometimes farther apart than normal. In other parts broad fibres may be seen, normal or nearly normal in aspect (Fig. 156) coursing among the fat cells, and accompanied by a smaller amount of fibrous tissue. Fibres occasionally present fatty degeneration, a longitudinal striation or fissuring, vitreous ("waxy") degeneration, or vacuolation, but these are rare. Some empty sarcolemma sheaths may be seen where the narrowing of the fibres is greatest. In muscles that still preserve some red tint, the amount of fat is less, and there is often a relatively larger amount of interstitial fibrous tissue. Very rarely, in some part of a muscle, there has been only wasting of the fibres, without the interstitial change, present elsewhere (Singer, in the triceps).

In some of these the interstitial tissue may be almost entirely fibrous, a few fat cells only being visible here and there. In such muscles it is common to find the fibres more damaged than in those in which the growth is partly fatty. It is chiefly in the latter that many normal fibres are seen. Muscular fibres larger than normal are seldom found after death, but such fibres have been seen in fragments removed during life by excision, or by a "harpoon-trocar;" it is possible that the increased size was due to a vital contraction under the mechanical stimulus involved in the extraction, but enlargement has sometimes seemed unquestionable (See note, "Muscular Hypertrophy").

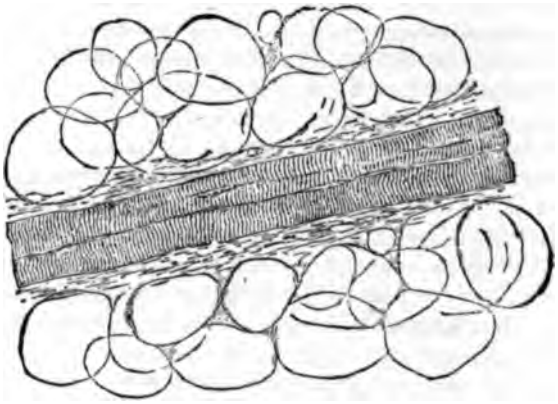

FIG. 156.—Gastrocnemius muscle; two nearly normal muscular fibres, accompanied by fibrous tissue, surrounded by fat cells.

The motor nerves, when examined, have been found normal. The condition of the sensory muscle-nerves (which terminate in the interstitial tissue in which the primary morbid process occurs) has not been ascertained in any instance. The spinal cord has been found perfectly normal in most cases in which it has been examined. In a few there have been slight and irregular degenerative changes. They have not been so marked in any other case as in one examined by Lockart Clarke and myself,* but the anterior grey matter was unaffected, and the changes were probably merely associated, and not the cause of the symptoms. Hæmorrhages have been occasionally found. Probably they have occurred late in life in the degenerated tissue. The neuroglial cells have been found increased in number, and the fibres of the white substance have been found irregularly changed, in a few cases, without any constancy in the seat of the alteration.

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* 'Med.-Chir. Trans.,' vol. lvii, p. 247. In this case the cervical and dorsal regions were normal, with the exception of here and there slight accumulations, at the bottom of the fissures, of products of degeneration, probably derived from the perivascular erosion common at all ages. At the last dorsal segment, however, there was an area of granular disintegration in the intermediate grey substance on each side, in front of the posterior vesicular tract. This part was unduly translucent for half a centimetre in vertical extent, and in the middle of this area the disintegration had produced an actual cavity, across which the fibres for the cerebellar tract ran unchanged.

Pathology.—The common integrity of the anterior grey matter of the cord, and especially of the motor nerve-cells, seems conclusive evidence that the disease of the muscles is not due to a primary lesion of the spinal cord. The slight irregular changes occasionally found are probably consecutive to the long inaction of the cord and deformity of the spine. Pseudo-hypertrophic paralysis is not therefore, as was at first thought, merely a form of spinal muscular atrophy with a special muscular change. The significance of the pathological anatomy is that the malady is a primary disease of the muscles, consisting in an overgrowth of the interstitial connective tissue, in which fat may or may not be deposited, and that the wasting of the muscular fibres is secondary to the interstitial growth. The indication of the conditions under which the disease occurs is that the disease is congenital, the result of a perverted tendency of growth, inherent in the embryo, and derived from the germ from which the embryo proceeds. In this connection it is instructive to note that there is one form of congenital tumour, the structure of which is almost exactly the same as that of the muscles in pseudo-hypertrophic paralysis. Fig. 157 might be a fragment of a muscle in this disease, but it is a section of a myolipoma, the congenital character of which is emphasised by the fact that it was attached to the conus medullaris of the spinal cord of a patient whose muscles were healthy. (The tumour is shown also in Fig. 163). It must have been due to the misplacement of some of the embryonal elements from which muscular

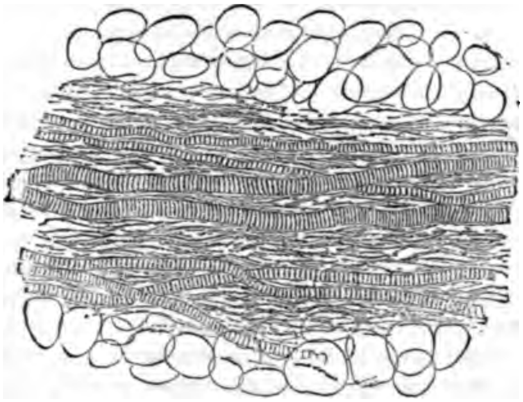

FIG. 157.—Section of a myolipoma which was attached to the spinal cord of a man suffering from locomotor ataxy, for comparison with Figs. 155 and 156.

tissue is developed, and it shows that, from such elements, the structural condition found in pseudo-hypertrophic paralysis may arise. The points of chief importance in the general pathology of the disease have been mentioned in the introductory remarks. The difficult question of the precise mechanism by which the muscular fibres suffer must be regarded as an open one. We may assume a defective vitality in them, but the conspicuous lesion is the growth of connective tissue, and by this the fibres are doubtless damaged, whether fatty tissue is formed or not. Indeed, the fibres seem to suffer more when there is only fibrous tissue than when there is the fatty deposit, although it is chiefly the fat that causes the enlargement of the muscles. There seems to be a tendency to the formation of fat in the early stage of the disease, and to its removal in the later stage, since in the former the muscles may often be observed to increase in size, and in the latter they become smaller, partly from some removal of fat and partly from the increasing atrophy of the muscular fibres. It is then that the greatest impairment of power occurs. In the cases in which the muscles are small from the first, the tendency to the deposit of fat seems slight, and the condition is an almost pure muscular sclerosis. The late shortening which occurs seems to be due to the contraction of the interstitial fibrous tissue, and it often coincides with the shrinkage from the removal of some of the fat, and the atrophy of the muscular fibres.

If it be true that the latissimus dorsi and lower half of the pectoralis are sometimes congenitally absent in pseudo-hypertrophic paralysis, the fact is quite consistent with the explanation of the nature of the disease given above. These two muscles stand perhaps lowest, in functional importance, among the muscles of the body, being used chiefly for the rare depression of the arm against a resistance (see p. 26). It is readily intelligible that a defect in the embryonal tissue of the muscular system should be quantitative as well as qualitative.

The loss of the knee-jerk is sufficiently explained by the lesion of the muscles. According to the theory that the irritability on which the jerk depends is due to a muscle-reflex action, the loss is readily intelligible, since the afferent impulse is due to the stimulation, by tension, of the sensory muscle-nerves, and these end (or begin) in the interstitial tissue, which is the seat of the primary morbid process in this disease. But the wasting of the muscular fibres must also be capable of abolishing the knee-jerk when the atrophy reaches a considerable degree, and it is also possible that the motor nerve-endings share this structural damage. On any theory of the nature of the knee-jerk, this will explain its loss. It is only when the muscular changes have attained a considerable degree that the loss occurs. The fact therefore does not, in itself, suggest any lesion of the spinal cord. If the malady is thus one of a morbid developmental tendency, it is not surprising that a tendency to analogous developmental diseases should be sometimes present in the nervous system, and that thus imbecility and epilepsy on the one hand, or albinism on the other, should be occasionally associated with the disease.

DIAGNOSIS.—The diagnosis of the disease is usually easy, if its characters are known. The peculiarity of gait and the mode of rising from the floor, the age of the patient, and the progressive character of the impairment, are in themselves sufficient to suggest the affection, and examination reveals enlargement and often contraction of the calf-muscles, and a change in the size of others, which confirm the diagnosis. The mode of rising is not absolutely pathognomonic, and it has misled even good observers; it is occasionally acquired in other diseases in which there is a gradual weakening of the extensors of the hip and knee. It is met with equally in the simple idiopathic atrophy, but is so rare from any other cause as to be of very great diagnostic suggestiveness.

Of the condition of the muscles, that which is most characteristic is the combination of enlargement of the infraspinatus with a wasting of the latissimus and lower part of the pectoralis. I pointed out some years ago* that this condition, which is seldom absent, is of very high diagnostic importance, and subsequent observations have fully confirmed the opinion. Next in importance is the enlargement of the calf-muscles, especially in combination with contracture that cannot be overcome. In general, the enlargement and diminution of neighbouring muscles is very significant, but it must be remembered that they may be gravely diseased, and yet of normal size, or from the first smaller than normal. In such cases they are often hard, and the distribution of the affection is the same as that of the double change in typical cases. Such cases illustrate the relation of this form and the simple atrophy described in the next section.

The disease with which confusion is most common is the so-called "congenital spastic paraplegia" (p. 441). Both diseases affect children; in both there are weakness of the legs and contraction of the calf-muscles, and in both the muscles are frequently large. The chief distinctions have been already mentioned. The most important are the preservation and excess of the knee-jerk in spastic paraplegia, the tendency to spasm of the legs,—the facts that the contracture is active and can be overcome, that the patient does not rise from the ground in the way peculiar to pseudo-hypertrophic paralysis,—and the opposite tendency of the two diseases. Spinal muscular atrophy is only likely to be confused with the simple atrophy.

Between the two forms of idiopathic atrophy, the distinction is scarcely one of diagnosis proper; it is rather a question of the category in which a case should be placed. The most important distinction is the freedom of the calf-muscles from enlargement in the atrophic form. The face is not affected in pseudo-hypertrophy as it so often is in idiopathic atrophy, except only in some intermediate instances. If more than one member of a family is affected, some of the sufferers will usually present characteristic symptoms of the special form, for it is remarkable with what constancy the two types generally remain distinct (see p. 403).

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* 'Pseudo-hypertrophic Muscular Paralysis,' London, 1879.

PROGNOSIS.—In the case of any child with pseudo-hypertrophic paralysis, the prognosis is most grave. It is almost certain that each year will bring increasing disability, and that the patient will not reach adult life. It is only when the disease develops late, and the symptoms do not become considerable until after twenty years of age, that there is a possibility that the disease may not attain its ultimate degree, but even in such cases this hope is seldom realised. In any case, and at any age, it is unlikely that the patient will live more than seven years after the power of standing is lost.

TREATMENT.—As a congenital developmental malady, pseudo-hypertrophic paralysis is one of those diseases in which medicine is necessarily powerless to cope with the essential elements of the process. As may be, therefore, expected, no drug has been found to exert an influence on the course of the affection, although such nervine tonics as phosphorus and arsenic have been thought sometimes to retard for a time the progress of the weakness. The stimulation of the muscles by electricity has been employed and advocated, but, however sedulously employed, no distinct result follows the use of either faradism or voltaism. It must be remembered, moreover, that electricity is a very feeble agent in stimulating the growth of muscular fibres, compared with the physiological stimulus of voluntary effort. Muscular exercise may reasonably be looked to in order to make up, in some degree, what is lacking in this disease, and does seem to have some influence in retarding the failure of power. It may perhaps induce further growth, or greater power in the muscular fibres that have not yet suffered, or actually supplement the defective trophic energy. When muscular exercise is stopped, there is certainly a quicker failure of strength. Hence it is desirable that the patient should carry out carefully planned gymnastic exercises, so arranged as to call into action the muscles that most need help. These, thoroughly persevered in, have seemed, more than any other means, to retard the disease. Although they have not in any case arrested it, the trouble that is necessary to arrange the method is certainly well spent. Rubbing and massage improve the circulation, and tend, especially when combined with passive movements, to lessen the tendency to muscular contraction and consequent deformities.

The influence of muscular exercise renders it very important to maintain locomotion as long as possible. The ability to stand and walk is generally lost, through the contraction of the calf-muscles, some time before the muscular weakness would take the patient off his feet. In such cases, tenotomy may restore the power of walking for some years, and when the contracture returns, its removal has, a second time, enabled walking to be resumed. The operation is thus distinctly beneficial, and should be performed as soon as the actual need for it arises, and division of the tendon is far better than any imperfect substitute.

During the later stages of the disease great care is required to preserve the patient from catarrh, which helps to excite the pulmonary mischief that so often ends life. Similar care is also needed during any intercurrent malady which the patient may contract.

Simple Idiopathic Muscular Atrophy.[*]

The cases in which there is no muscular enlargement, in which wasting is manifest from the first in the size of the muscles, are much more rare than the pseudo-hypertrophic disease. They belong to several types, more or less distinct, of which the most common is that to which Erb has given the name of "juvenile," and of which most instances are probably the pseudo-hypertrophic disease without muscular enlargement; others are more special since the lower leg muscles have suffered but little. A more striking variety is that which is characterised by the affection of the muscles of the face in addition to those of the shoulder girths—the "facio-scapulo-humeral type" of Landouzy and Déjérine. In a third variety the affection begins in the legs and is peculiarly slow in course. Some other types may ultimately be differentiated, but it is important to recognise the fact that even those that we can now distinguish, as the most pronounced in their features, do not keep entirely distinct. They are connected, by intermediate forms, not only with each other, but even with the pseudo-hypertrophic disease.[†] But these are very rare; as a rule the two latter types remain distinct in the families in which they occur, and this is the justification for their separate description. In those families in which simple muscular atrophy occurs, however numerous the cases, however different the distribution of the disease, and however various the ages at which it begins, cases rarely present the distinctive characters of pseudo-hypertrophic paralysis.

[*] The most important writings on the subject (besides those specially quoted) are those of Duchenne in 'Électrisation Localisé' (p. 60 of Poore's translation, published by the New Sydenham Society); Barsickow, 'Inaug. Dissert.,' Halle, 1872; Leyden, 'Klin. d. Ruckenm. Krank.,' Bd. ii, p. 525; Möbius, "Hered. Nervenk.," Volkmann's 'Klin. Vorträge,' No. 171; Landouzy and Déjérine, 'Revue de Méd.,' 1885, pp. 81 and 251; Marie and Guinon (a series of cases observed at the Salpêtrière), ib., 1885; Sachs, 'New York Med. Journ.,' Dec. 15th, 1888; Hitzig, 'Berlin klin. Wochenschr.,' 1888; Singer, 'Zeitscn. f. Heilk.,' Bd. viii. Especially valuable are the papers by Erb, 'Deut. Archiv f. klin. Med.,' Bd. xxxiv, 1884, and 'Neurol. Centralbl.,' July 1st, 1886, with numerous later writings. Bibliographical references, &c., will be found also in Tooth's 'Thesis on the Peroneal Type,' and in my Lecture on Pseudo-hypertrophic Paralysis (1879), and in Sach's article (loc. cit.).

[†] *E.g.*, case described and collected by Marie and Guinon (loc. cit.), connecting the pseudo-hypertrophic form with both the juvenile and the facial.

There is less constant separation between the simple "juvenile" and the facial forms. Although they often keep distinct, the affection of the face has been absent in a few cases in a family in which it was the first part to be affected in most sufferers. Thus a man with simple atrophy, beginning at fourteen in the shoulder- and thigh-muscles, whose face was unaffected, had a daughter who began to suffer at eleven, and in whom the face, scapular, and arm muscles were involved.* The peculiar "peroneal type" is not included in this outline, and the statements here made do not apply to it.

CAUSES.—We are able to trace no cause beyond the congenital tendency, already considered, shown by the occurrence of many cases in the same family, and in more than one generation. In one remarkable series recorded by Barsickow, twenty-four cases were distributed through five generations, and the disease was also traced through five generations in a group described by Landouzy and Déjérine. It is very rare for the disease to be confined to one generation, far more rare than for pseudo-hypertrophic paralysis to be so confined. But, as in the case of most congenital hereditary diseases, cases that are apparently isolated are occasionally met with. Such isolated cases are rare,—more so, probably, than in the pseudo-hypertrophic form; although wider observation may show that they are more frequent than we now suspect. In the families of the patients shown in Figs. 158–61 and 162 no example of analogous disease could be heard of.

Both sexes suffer; in a few families, females chiefly; in others, males; in most, both have been affected. The age at which the disease first manifests itself is extremely variable. It may begin as early as two† or three‡ and as late as sixty years. But the onset is seldom during childhood; in the majority of cases the disease shows itself between fifteen and thirty-five; that is, during the later period of growth and the early period of adult life. Even in the same family, the variations may be extreme; in that described by Barsickow the date of the onset of seventeen cases was known, and was as follows:—in one at 12; in four between 15 and 20; in seven between 20 and 30; in three between 30 and 40; in two after 40. A woman, aged fifty-two, began to suffer at 30, but her son at 3 years of age.§ Sex has no influence on the date of onset, nor, as a rule, can any relation be traced between the date and the place at which the wasting begins. When the wasting begins in the face, the disease more frequently commences in childhood than when the first symptoms are in the limbs, but, in some instances, the atrophy has commenced in the face late in life, and this in the same family in which other sufferers have been young. Thus in the sexual proclivity, and in the date of onset, there is a marked difference between this form and pseudo-hypertrophy; the latter showing a stronger tendency to affect males, and to manifest itself in childhood. The "juvenile" form, as a rule, presents less difference.

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* Troisier and Guinon, 'Rev. de Méd.,' 1889, p. 48. See also Singer, 'Zeit. f. Heilk.,' viii, p. 229.

† Kreske, 'Munch. Med. Wochenschr.,' 1886.

‡ Landouzy and Déjérine, loc. cit.

§ Ibid.

As a rule, no direct exciting cause can be traced. In a few instances the onset has succeeded some other morbid process, such as chlorosis, acute disease, or rheumatic affections due to exposure to cold; and the depression of general health resulting from these may have determined the time of onset, but is not likely to have done more. In other individuals of the same families the disease has developed without the aid of any exciting influence.

SYMPTOMS.—The onset is always gradual. Weakness and wasting come on together, and are noticed simultaneously, unless the commencing atrophy is concealed by subcutaneous fat. The atrophy generally begins in the upper arm and shoulder-muscles, having, in the most common "juvenile" form, a similar distribution in these parts to that of pseudo-hypertrophic paralysis. In the facial form, however, this part usually suffers first, as in the lad figured on p. 406. In rare cases the wasting not only begins in the legs, but remains limited or almost limited to them. In the part first affected the disease slowly increases, and thence it usually spreads. The onset may be symmetrical on the two sides, or one side may suffer some time before the other.

Of the arm-muscles, the weakness and wasting are noticed first in the biceps and triceps, and with these the supinator longus often suffers. But examination generally shows that the lower part of the pectoralis and latissimus dorsi are greatly wasted, a loss of which the patient may be little aware, on account of the relative unimportance of these muscles. Sometimes the upper part of the pectoralis, and even the pectoralis minor, are also affected (Fig. 159). The tendency to atrophy of the lower part of the pectoralis and latissimus is a character which, as already stated, is common to this disease and pseudo-hypertrophic paralysis, but not invariable in either. The deltoids are rarely involved; in many cases they are normal; sometimes they have been thought to be unduly large; in a few instances they have been wasted (Fig. 162). The serratus magnus is often affected (Fig. 161), but may escape, even in a severe case. The supraspinatus and infraspinatus may also suffer, but are often normal or even enlarged; the trapezius and rhomboids have been affected in many cases, sometimes much atrophied (Fig. 161), especially in the "juvenile" form.

The forearm-muscles generally escape, with the exception of the supinator longus. Occasionally there has been some weakness of the long extensors or flexors of the fingers, with or without slight visible wasting. In the case mentioned on the next page, the extensors of the phalanges of the thumb were involved on the left, and the radial extensor of the wrist on the right side. Rarely the forearm-muscles have been much atrophied. In several instances there has been some atrophy of the small muscles of the hands, the thenar and interosseal muscles, or the interossei only, as in the case of Landouzy and Déjerine; but in the majority the integrity of these muscles is a marked feature of the disease, and a contrast to the spinal form.

The affection of the face is peculiar. There is commonly a failure of the zygomatic muscles, and, in consequence, a loss of the naso-labial furrow, and a curious alteration in the smile; instead of the angles of the mouth being drawn outwards and upwards, they are moved upwards by the elevators of the upper lip and angle of the mouth. The orbicularis oris is also affected, and, in consequence, the lips are habitually separated, the lower lip projects, the patient cannot "pout," and the articulation of labials is imperfect. The face has a dull expression and the aspect is very peculiar; it has been termed, by Landouzy and Déjérine, the "myopathic face." In one case, observed by them, the face was unequally involved on the two sides. Rarely (as in Fig. 159) the frontales have been involved, and the orbicularis palpebrarum has been weak. Weakness of the eyelids was supposed to be the cause of distinct exophthalmos present in one woman.* In the case described by Kreske, inability to close the eyes was noted at three, and at ten the paralysis of the face was absolute. Wasting of the muscles may be indistinct, because the contour of the face is only to a slight degree influenced by the substance of the muscles. The projecting lower lip may, indeed, appear to be thicker than normal. In many cases the buccinators have been affected, in some instances they have been normal, and then have drawn out the angles of the mouth in smiling. The tongue has been always unaffected, and so also have the pharynx, larynx, muscles of mastication, and the eyeball-muscles.†

The muscles of the spine have sometimes been normal, sometimes they have been considerably atrophied.* The intercostals are often affected in the later stages, but rarely in extreme degree. The diaphragm also sometimes suffers, as in the case shown in Fig. 158. The abdominal muscles have been involved in only a few instances. In the legs the muscles most commonly affected are the flexors of the hip, the extensors of the knee, and less frequently the glutei. The muscles below the knee have escaped in many cases; when they have suffered, the atrophy has often been general. When the peroneal and anterior tibial muscles have been specially affected, as in the case shown in Fig. 162, the case has generally been of the peculiar "peroneal" type to be presently mentioned, although it is possible that some instances of the kind are on the borders of the group now under consideration.

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* Landouzy and Déjérine.

† In a singular case under my care some years ago, an affection of the facial muscles, similar to that of idiopathic muscular atrophy, was associated with paralysis of ocular muscles. The patient was a girl twenty-seven years of age; there were no indications of syphilis, nor could any history of muscular atrophy in the family be ascertained. The ocular palsy commenced gradually at twenty-four, and increased until the movements of both eyes upwards, of the left eye inwards, and the right outwards, were lost, and all other movements were weakened. The eyelids drooped slightly; the internal ocular muscles were normal. The affection of the face followed that of the eyes; the zygomatic muscles were powerless, so that the smile consisted only in elevation of the upper lip; the orbicularis was weak. The palate, pharynx, and larynx were normal. The arms became feeble, and the flexors of the hips almost powerless. There was no visible change in the nutrition or electrical irritability of the muscles; the knee-jerk was normal. If the case was central, as it appeared to be, the peculiar affection of the lips and zygomatics is not confined to idiopathic muscular atrophy. If the disease was muscular, the eyeball-muscles do not invariably escape. With this case may be noted one recorded by Oppenheim ('Charité Annalen,' xiii) as the "juvenile" form, with derangement of the lateral movement of the eyes, nystagmus, and some laryngeal palsy. The shoulder- and thigh-muscles suffered chiefly, but, below the knee, the peronei—a noteworthy aberration from the type.

The electric irritability of the affected muscles is usually lessened in proportion to the wasting, and equally to both currents. Indeed, the diminution seems sometimes out of proportion to the wasting, and great when the atrophy is slight. There is no trace of degenerative reaction, and there is not even the longer persistence of voltaic than of faradic irritability which occurs in the more chronic cases of spinal atrophy. Fibrillation is almost always absent, but not quite invariably; hence its absence cannot be made distinctive, still less its presence. Myotatic irritability is lessened or lost; it is never increased. Some shortening of muscles has been occasionally noted, frequently in the calf-muscles, rarely in the biceps.

All other functions of the nervous system are unaffected. Sensibility is normal. In a few cases transient rheumatoid pains have accompanied a rapid development of the disease in the arms, but in the vast majority of cases the disease is painless. The sphincters are unaffected, and there is no tendency to trophic or vaso-motor disturbance.

As the muscular atrophy progresses, the form of the affected parts becomes changed just as in spinal atrophy. Deformities may also occur, chiefly from the shortening of less affected muscles, but these rarely reach the considerable degree common in other forms of muscular wasting. Lordosis occurs in the upright posture, and is probably due to the same mechanism as in pseudo-hypertrophic paralysis, ceasing, as in that disease, when the patient sits (compare Figs. 158 and 161). When the muscles of the lower leg are involved, talipes may develop.

The course and duration of the disease are exceedingly variable. The atrophy may remain limited to the part in which it begins. The face alone has been affected in some members of a family, although in other members the limbs subsequently suffered. In cases in which the wasting spreads, years may intervene before the extension takes place. In one case, for instance, the right arm became affected at nineteen, the left at twenty-five, the legs at thirty. In another the arms began to waste at thirty-five, power of standing was only lost at

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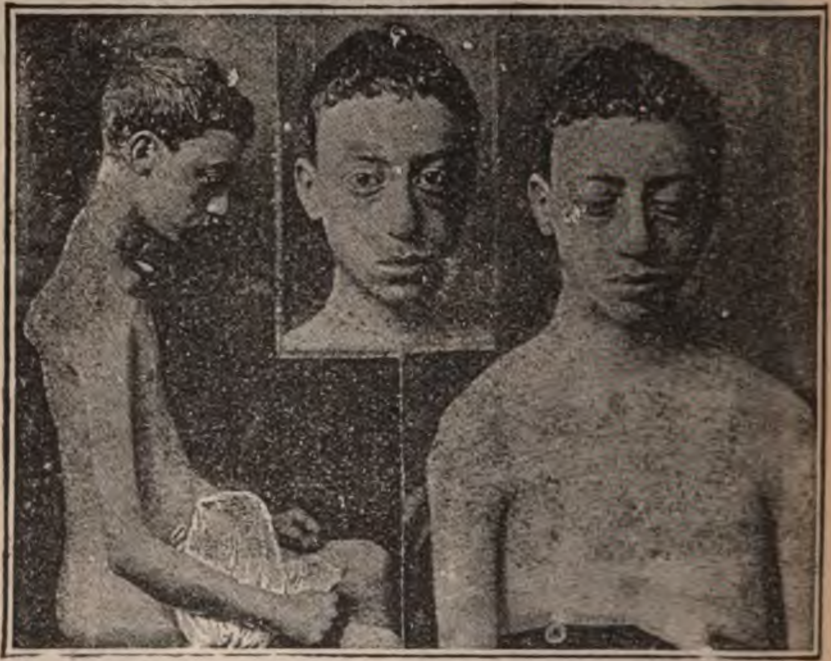
* Atrophy began in the back-muscles at forty-four in a case recorded by Musso ('Riv. Clin.,' June, 1887), and twelve years later had involved also the legs. The patient's mother, two brothers, sister, and maternal uncle suffered in the same way at the same age.

FIG. 158. FIG. 159. FIG. 160.



FIGS. 158 TO 161.—Idiopathic muscular atrophy affecting the face. Fig. 158 shows the wasting of the deltoid upper-arm muscles and supinator longus (while the forearm-muscles are not wasted), and the rotation of the scapula from the loss of the trapezius and rhomboids. Fig. 159 shows the habitual appearance of the face, and Fig. 160 the greatest possible movement of the facial muscles in closing the eyes and smiling, and also the wasting of the deltoids and pectorals. The general distribution of the atrophy and projection of the scapulæ are seen in Fig. 161.*

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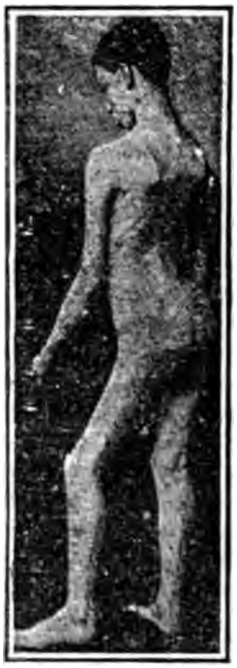
* The patient was a lad sixteen years of age. The following is an abstract of the notes of his case. In his family no history of any similar affection can be ascertained. There is conclusive evidence that he had a hard chancre at the age of three, followed by secondary symptoms. The muscular wasting began gradually during childhood; even when very young there was little movement in the face, and that ceased at the age of six or seven. Walking became difficult about three years ago. The face is almost motionless: an effort to smile causes only a just perceptible movement of the left cheek, the amount of which is fairly indicated by the difference between Figs. 159 and 160. It is accompanied by a distinct movement of the ears. The lips are full and can be brought together, but not shortened. The eyelids cannot be made to meet, even with a strong effort, being then ⅛ in. apart. Forehead absolutely motionless. Eyeballs prominent, movements normal. Masseters, tongue, pharynx, and larynx unaffected. In the neck the sterno-mastoids are very small and

fifty-five, and the patient lived till seventy-five. Even when the malady begins in childhood its progress is sometimes very slow. Thus in one case the affection of the face was first noticed at five, at twelve the arms began to suffer, and a few years later the flexors of the hip became weak, but the patient was still able to walk at the age of forty. The disease may increase until development is over and then remain stationary, as in a case in which, at forty-four, there had been no change since the age of twenty. The patient's daughter was more severely affected.* On the other hand, the progress of the disease may be more rapid and uniform, and the atrophy may reach its widest distribution in eight or ten years. In most cases, even of severe type, the wasting does not become universal, but remains limited to the muscles mentioned above, but occasionally hardly any muscles of the body may escape.

FIG. 161.



The duration of the disease varies from ten to fifty years. Death has never occurred as the direct result of the malady. In the cases of most severe degree and rapid course, the patient has usually died from phthisis, probably related to the deficient breathing power, just as in the sufferers from pseudo-hypertrophic paralysis. In most cases, however, death has been due to other maladies, and has not been in any degree the consequence of the muscular disease.

PATHOLOGICAL ANATOMY.—In general, the condition of the muscular fibres resembles that met with in pseudo-hypertrophic para-

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feeble, the omo-hyoids large and strong. The following muscles seem quite gone—trapezius (except, perhaps, a little of the middle part of the right), rhomboids, deltoid pectorales, latissimus dorsi, serrati, biceps, brachialis, triceps, supinator longus. The lev. ang. scapulæ, supra- and infra-spinatus are unaffected; the latter is large but probably only hypertrophied from over-use. The supinator longus is feeble on the left, powerless on the right side. The extensors of the phalanges of the thumb are lost on the left side, and the extensor carpi radialis on the right. All the other forearm- and hand-muscles appear to be unaffected. The erectors of the spine are small and feeble; intercostals strong, diaphragm powerless, abdominal muscles weak. In the legs the glutei and flexors of the knees seem normal, the flexors of the hips weak, extensors of the knees small and feeble, muscles of the lower leg rather small and feeble, but only the peronei are powerless, right-angled contracture of ankle-joint with tendency to varus. Knee-jerk absent. Circumference (in inches) of upper arms (middle), R. 5$\frac{15}{16}$, L. 5¼; of forearm (maximum), each 8; of thighs (minimum 2½ inches above patella), R. 10¼, L. 9¾; calves (maximum), R. 10¾, L. 11. Electric irritability much lowered in all affected muscles to each current; in most, no contraction can be obtained. Sensation, the sphincters, and the visceral functions are unaffected.

* Landouzy and Déjérine, loc. cit.

lysis, the increase of interstitial tissue being wanting except in those cases that really belong to this form, but in which the characteristic enlargement is absent, because no fat cells are developed in the tissue. A multiplication of nuclei is, however, sometimes seen. The chief change is sometimes a simple narrowing of the fibres, with an ultimate disappearance of the transverse striation. Degenerative changes are frequently met with, indistinct striation or granular, fatty, or "waxy" transformation being seen here and there, alike in narrowed or enlarged fibres, and in those that retain their normal calibre; occasionally fibres present longitudinal striation, fissuring, or vacuolation; but in other cases, or in some muscles (especially in the "juvenile" form), a remarkable increase has been found in the size of fibres in excised fragments,* even of muscles that are below the normal size. It seems to show pathological enlargement, although allowance has seldom been made for the influence of excision, mentioned in the chapter on Hypertrophy.

The statements made regarding the spinal cord in the pseudo-hypertrophic form, are strictly applicable also to this variety. It is normal as a rule, to which exceptions are so rare that they can have no relation to the malady, save that of secondary indirect consequences. The nerves also are normal, if the cases are eliminated in which the symptoms indicate the peroneal form.

Thus the facts ascertained regarding this type do not suggest any special addition to the general conclusions intimated in the introductory paragraphs. The affection appears to be the result of a primary developmental tendency, involving the muscular tissue only, and cannot be regarded as a secondary result of the interstitial changes, except in a trifling, occasional, and unimportant degree. Still less is it a consequence of changes in the spinal cord. If it is in any measure the result of failure in the nutrition of the terminal structures of the nerve-fibres (by which the nervous and muscular tissues are connected, and which is perhaps to be regarded as intermediate between the two), the fact has yet to be demonstrated, and its significance ascertained.

DIAGNOSIS.—The two most important diagnostic indications are the affection of more than one member of the same family, and the onset of the disease before adult life is reached. The former is practically conclusive; and the latter should always suggest the probable idiopathic nature of the case. In isolated cases the diagnostic difficulty is much greater, and no indication is actually conclusive, except, perhaps, the affection of the face. Commencement during childhood is also of great weight. It is most unlikely that progressive muscular atrophy, beginning under ten, is of spinal origin, but I have known such atrophy, certainly spinal, to commence at the age of fourteen. The peculiar affection of the zygomatic muscles is very characteristic, but we cannot yet say that it is pathognomonic (see note on p. 405), and the affection of the lips must be carefully distinguished from that due to the bulbar palsy so commonly associated with spinal atrophy. Of the distribution of the wasting, that of the latissimus and lower half of the pectoralis, and the escape of the hand-muscles, are the most important characteristics, although they are not, by themselves, conclusive, since they are sometimes met with in spinal atrophy.

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* See especially Schulze, 'Ueber den mit Hypertrophie verbundenen Muskelschwund,' Wiesbaden, 1886, and Hitzig, loc. cit.

But the most important distinctions between spinal and idiopathic atrophy, whenever they are available, depend upon the symptoms of a lesion of the cord, or of the bulbar nuclei. Especially significant is the excess of the knee-jerk which results from lateral sclerosis, a conclusive indication of spinal disease, rendering the spinal origin of the atrophy all but certain. The distinction from the pseudo-hypertrophic form has been already considered; that from the peroneal type will be presently mentioned.

PROGNOSIS.—The extreme variations in the course of the disease render the prognosis in any individual case both uncertain and difficult to formulate. Speaking generally, chronicity favours arrest, and the later the symptoms appear, the slower will be their progress. The prognosis is distinctly less grave in the simple atrophy than in the "juvenile" atrophic variety of the pseudo-hypertrophic disease, and in a considerable number of cases, perhaps one half, the malady has not appeared to shorten life. Even in cases that begin during youth it is therefore possible that the patient may reach old age. It seems also to be better in the cases that do not involve the facial muscles.

TREATMENT.—It is not yet clear that idiopathic muscular atrophy can be influenced by treatment in any considerable degree. Most published records of cases are almost silent on the subject, and the disease is so rare that few individuals have an opportunity of forming an opinion. It might be assumed that the essential cause of the disease, a congenital tendency, withdraws it from the range of therapeutics, but the course of the malady is very different from that of some other diseases which own a similar cause. The extreme variations in the date of onset, the fact that the disease may not be manifested until late in life, and the long period that may intervene between its onset and extension, all suggest that other influences co-operate with the congenital tendency in determining the development of the malady. It is quite possible, therefore, that the first inference from the history of the disease may not be altogether correct, although it must be admitted that we have not as yet any evidence that the disease is susceptible of influence from drugs. Electrical treatment and massage have been thought to do good and even to produce arrest (Erb), but the variable tendency of the malady renders the evidence of arrest insufficient. If voluntary exercise is practicable, this is a far more efficient stimulus to muscular growth than any electrical application, and it is probable that such exercise, carefully persevered in, may do something to prevent the occurrence of the malady in those predisposed to it, and even to retard its progress in those who are already attacked. In this connection it is noteworthy that very few of the sufferers have been engaged in occupations that involve active muscular exertion. Over-exertion should, however, be carefully avoided. The general health should be attended to, and any defect removed as speedily as possible, both in those who suffer and in those who are related to sufferers, since, as we have seen, depressing influences may apparently excite the development of the disease, and it is therefore reasonable to suppose that they may accelerate its course, and lessen any tendency there may be for the morbid process to become stationary.

THE PERONEAL TYPE OF FAMILY AMYOTROPHY.

The name "peroneal type" has become current as the most convenient designation for the variety of muscular atrophy, occurring in early life, to which it was applied by Dr. Howard Tooth in a Thesis published in 1886.* This was the means of calling general attention to the form, of which, however, an account had been published shortly before by Charcot and Marie,† and many cases had been previously described, in most instances without recognition of their special character.‡ The cases of this form present certain resemblances to the idiopathic atrophies just described—resemblances sufficiently important to have led to the description of these cases in association with the primary myopathy, embracing, as they do, the age of the sufferers, the occurrence of the disease in members of the same family, its gradual onset, and its very slow but progressive course. Yet this position must be regarded as provisional only, and probably erroneous. The distribution of the wasting involves a constant difference from the idiopathic muscular affections, and some of the cases, otherwise inseparable from the rest, present features that indicate neuritis. The disease may have, as an antecedent, an acute specific disease—a circumstance that has the same significance. The facts ascertained concerning this form must, therefore, be carefully excluded from our generalisation regarding the idiopathic atrophy.

In this form, males suffer about twice as frequently as females. It generally begins in the second half of childhood, and very seldom after twenty, although cases have been described beginning as late as forty. It is occasionally hereditary, and still more frequently collaterals suffer, several brothers and sisters being affected. The wasting is first apparent in the extensor longus pollicis, or extensor communis digitorum, or in the peronei muscles. According to Tooth, it very often begins in the latter, and occasionally in the gastrocnemius, but it is probable that it occurs simultaneously, or even earlier, in the small muscles of the foot, where it readily escapes observation. Such early atrophy in the feet has, indeed, been actually found in some cases that came under observation at an early stage.* The calf-muscles suffer subsequently, and still later those of the thigh, especially the vastus internus. The unequal affection of the muscles of the leg causes the early development of club-foot, which, especially in children, becomes a characteristic symptom.† The arms are only invaded, in most cases, some years after the legs, so slow is the course of the disease. The intrinsic muscles of the hands (thenar, hypothenar, and interosseal muscles) are first attacked, and subsequently those of the forearm; sometimes the extensors and sometimes the flexors suffer first and most; while the supinator longus remains normal, and so do the muscles of the shoulder, neck, back, and face. The atrophy is often symmetrical, but occasionally the muscles on one side waste first and most. The claw-like deformity of the fingers may result from the affection of the intrinsic muscles of the hand. This is so rare in early life from any other cause, that its significance is considerable, and almost distinctive when combined with the deformity of the feet just mentioned. The case shown in Fig. 162 was probably an example of this type.‡

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* 'The Peroneal Type of Progressive Muscular Atrophy,' London, Lewis, 1886.

† 'Revue de Méd,' 1886, p. 97.

‡ By Friedreich, Eichhorst, Oppenheimer, Ormerod, and others. A table of thirty collected cases (some doubtful) is given by Tooth.

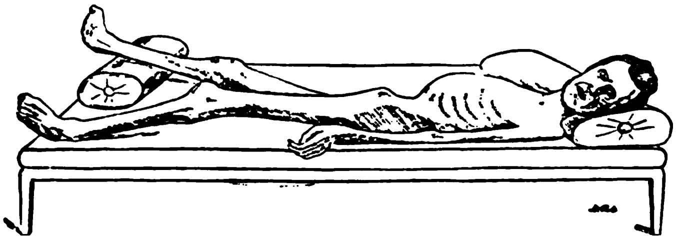

FIG. 162.—Advanced muscular atrophy, probably of the "peroneal type." (Drawn by Dr. Spencer, from a photograph.)

The affected muscles (especially in the hand) occasionally present slight fibrillation. Their electric irritability is diminished sooner, and in greater degree, than in the ordinary idiopathic atrophy, while a still greater difference is presented by the fact that faradic irritability may early become extinct, and that there may be a distinct reaction of degeneration. In connection with this, should be noted the important fact that cutaneous sensibility, though often normal, is sometimes impaired or lost, especially over the region in which the atrophy is greatest, or upon the soles of the feet. Pains occur in some cases, and so also do spontaneous spasmodic contractions, especially in the muscles of the thigh. The muscle-reflex action is lessened or lost in the affected regions, but there may be extensive atrophy below the knees, without loss of the knee-jerk, if the thigh-muscles are preserved. Cutaneous reflex action presents considerable variations, and is preserved more often than would be expected. Vaso-motor disturbance sometimes occurs (Sachs).

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* Hoffmann, 'Arch. f. Psych.,' xx, p. 660; Joffroy, 'Gaz. Hebd.,' 1886, No. 18.

† See Sachs, 'Brain,' Winter part, 1889.

‡ It was given in the last edition as an example of simple atrophy. The patient was a man aged twenty-seven at the time of his death (in University College Hospital). No history of any analogous case in his family could be ascertained. The affection began at the age of fourteen, when his feet began to turn in so that he walked on the outer side of the foot, and soon he noticed gradual wasting of the legs, which slowly progressed, and about the age of twenty-four the arms also began to suffer. When first seen, a few months before his death, the muscular atrophy was universal, and the subcutaneous fat had also disappeared. Even the hands were greatly wasted; there was a hollow in the position of the thenar eminence, just as in progressive muscular atrophy, and the interosseal muscles were also greatly wasted. There was slight fibrillation. The intercostals were paralysed. The wasting of the legs was extreme; the maximum circumference of the calf was

The nature of these cases has still to be demonstrated. Their very slow course, the time of life at which they begin, and the affection of several members of the same family, are features analogous to the idiopathic atrophy, but a marked contrast to this is presented by the tendency of the malady to succeed an acute specific disease, measles, and this also may appear as a family tendency. This feature resembles the causation of multiple neuritis, and the electrical reaction of degeneration is conclusive proof of preponderant changes in the motor nerves, while anæsthesia demonstrates that, in some cases, the sensory fibres are also affected. In such cases, a peripheral neuritis, motor or total, of extremely chronic course and peculiar origin, must be recognised as the only possible lesion, and it was actually found, in one case, by Friedreich, and in another by Gombault. If this is the cause of the symptoms in some instances, the question arises whether it is not the lesion in all the cases of this peroneal type. Such a lesion is conceivable; the degenerative form of neuritis presents every degree of chronicity, and may be limited to the motor fibres and to certain muscles. This pathology was advocated, as the probable one, by Tooth, who pointed out that it would also enable us to understand the occurrence of fibrillation, which is extremely rare in the idiopathic atrophies. It is, indeed, asserted by Hoffmann that not only are the peripheral nerve-fibres the seat of a primary degeneration, but that this change ascends the nerves to the anterior and posterior roots, and involves, in a secondary manner, the elements of the spinal cord, especially the fibres of the posterior median columns (which continue the path from the muscles) and the motor cells of the anterior cornua. But we do not know how far the premature

only seven inches; the feet were everted, the sole hollowed, and the toes flexed. The patient died from diarrhœa. The muscles were found to be small and pale. The chief microscopical change was intense granular and fatty degeneration of the fibres. A very few normal fibres were seen in some muscles. There was no increase of interstitial tissue. Microscopical examination revealed no morbid changes in the spinal cord. The nerves, unfortunately, were not examined.

failure of nutrition is a secondary process, transmitted from below, or how far it is the result of a defect in vital energy, shared by the central as well as the peripheral nerve elements.

The intimate relation between the motor nerves and the muscular fibres should also be kept in view in this connection. There may be a morbid tendency in the nerves similar to that of the muscles, including a liability to early slow degeneration, analogous to that which the muscles present, and equally prone to occur in several members of a family. But it is desirable that the neuritic nature of the affection should be placed on a broader basis of ascertained fact before it is allowed expression in the name given to the disease.

It should also be remembered as possible that the atrophy may be idiopathic in some cases, while in others it may be secondary to such a neuritis as above mentioned. There is nothing difficult, or even anomalous, in the conception of a congenital tendency to premature failure of nutrition in the peripheral nerve fibres, like that which, in the pyramidal fibres, seems to underlie "hereditary ataxy," or which, on the other hand, in the muscles, is the apparent cause of idiopathic atrophy. Further, if the lesion is really neuritic, we can understand that an acute specific disease like measles should occasionally be its excitant, although its occurrence in families shows that a congenital tendency must be called into activity by the virus, and the cases must be distinguished from those in which a pure neuritis is induced by the influence of the poison of the specific disease. An acute specific disease, the virus of which is known to be capable of inducing the degeneration of "parenchymatous" neuritis, may readily excite this state in each of several members of the same family, if a congenital tendency to it exists in them. It may readily evoke that which it could, even alone, produce. According to this theory, a tendency to nerve degeneration, acting alone, may lead to the early failure of nutrition of the nerves, while the wasting of their muscular fibres may be either a consequence or a concomitant, in part or altogether. Thus, the muscles may atrophy because the nerves have degenerated, or because they also have a tendency to atrophy, or from both causes combined. Nerve and muscle may alike share the congenital imperfection in some cases, while in others its primary incidence is on the nerves alone, the muscles suffering in consequence of the changes in the nerves. The congenital association may sometimes be even more extensive, and a tendency to the early failure of structures in the central nervous system may be conjoined, giving rise to cases of complex aspect and character.

It is especially important to note, in this connection, the occurrence of cases that occupy an intermediate position, and combine the characters of this and the myopathic types. Such combined cases are usually partial only, but such partial cases, when combined, ultimately cover the whole intermediate ground. For instance, the condition of the legs in the "peroneal type" has coexisted with that of the arms in the "juvenile form" in several recorded cases.*

Thus, the possibility must be recognised that the cases of this type vary, even more than their characters suggest, in the degree in which the morbid tendency acts upon nerve or muscle, or both, as well as the degree in which its activity is spontaneous or induced. Hence, the cases may here be only partly out of place, some perhaps not at all; and their position, as forms of "neuritis," might be equally open to exception.

The progressive character of the cases of this type is commonly pronounced, and, even if slow, it renders the prognosis gloomy, and the scope for treatment small. So far as anything can be done, it is by the measures already mentioned in the account of the treatment of the maladies to which the cases approximate, the primary disease of the nerves on the one hand, and of the muscles on the other.

MUSCULAR HYPERTROPHY.

The occurrence of a true hypertrophy of muscles, as a condition of disease, is exceedingly rare. In most cases in which the muscles are enlarged, the increase in size is due to a growth of interstitial tissue, fatty or fibrous, such as has been described in the chapter on pseudo-hypertrophic paralysis. We have seen that muscular fibres larger than normal have been described in that disease, and also in some cases of simple atrophy. When this increase in size has been met with only in excised fragments, it is possible that the condition may have been due to a vital contraction excited by the process of excision, and such evidence of hypertrophy is certainly inadequate;† only when it is found after death, can its occurrence be regarded as beyond question. Nevertheless, true hypertrophy of fibres is a change that may be expected to be occasionally met with in cases that are due to a congenital tendency, and its presence, even in muscles that are the seat of atrophy, need, therefore, excite no surprise. It was met with by

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* *E.g.* by Hoffmann, 'Berlin. klin. Wochenschr,' 1887, No. 22; and Eisenlohr, 'Neur. Centralbl.,' 1889, p. 565.

† Since the above was written, the doubt there expressed has been confirmed by an observation made for me by Dr. H. R. Spencer. A fragment of the gastrocnemius of an amputated leg was excised immediately after the amputation, and the fibres were compared with those of the muscle twenty-one hours later. In order to ascertain more definitely whether a vital contraction persisted as an apparent increase in size, a part of the excised fragment was separated and faradised. The average size of the fibres in the muscle was $\frac{1}{760}$ inch, in the fragment excised $\frac{1}{530}$ inch, in the fragment faradised $\frac{1}{517}$ inch. Since the division of the nerve during the amputation may have caused some contraction, it is possible that the difference in size produced by excision may be even greater than these figures represent. [I leave the above note unchanged because, since it appeared in the first edition, the observation and doubt have been fully confirmed by various observers, *e.g.* by Oppenheim and Siemerling, 'Med. Centralbl.,' 1889, No. 39.]

Bruck in a remarkable case of general muscular enlargement in an idiotic child, in whom it apparently developed after birth. The affection first showed itself in the tongue, but the muscles presented spasmodic contractions, and the case, like most of the kind, was evidently anomalous in its features. Muscular hypertrophy has been found in the singular malady described in the next section (Thomsen's disease). It has also been met with, in very rare instances, as a widespread or partial condition, usually associated, strange to say, with either diminished power or with a morbid readiness of fatigue.* The muscles most frequently affected have been those of the shoulder and upper arm, or of the thigh and calf, on one side or on both. It has also been observed in the glutei, spinal muscles, and trapezii. The causes are obscure, but it has been ascribed to over-exertion. The diameter of the fibres has been increased to double the normal, a maximum of $\frac{1}{200}$th inch having been met with (Eulenberg), whereas the normal maximum may be taken as $\frac{1}{400}$th inch. An increase of the nuclei has been observed, without any overgrowth of the interstitial tissue.

The condition is manifested by an increase in the size of the muscles, which are also firm. The circumference of the limb is greater than normal, and when the change is unilateral, the difference between the limbs of the two sides may be very striking. The maximum circumference of the calf has been as much as seventeen inches. The muscles are soon exhausted, and have been weak in some cases, while in others there has been abnormal strength for brief exertion. The electrical, mechanical, and myotatic irritability of the muscles has usually been found unaltered.

The disease may be suspected if a marked increase in size in an adult is accompanied by impaired power of sustained exertion; if the muscles are firm, and the patient does not manifest other indications of pseudo-hypertrophic paralysis. The diagnosis can, however, only be made with certainty by the microscopical examination of an excised fragment, and even then subject to the reservation mentioned on the previous page. The meagre facts regarding the course of the disease suggest that it usually persists without getting either better or worse. Treatment appears to have but little influence upon it.

A singular case has been reported by Eulenberg,† which differs in many particulars from the form of disease above described, and illustrates the complex relations of enlargement of the muscular fibres and its connection with degenerative processes. A man, aged thirty-six, presented an enormous enlargement of the muscles of the left leg, which were soft and flabby and weak, with lowered irritability. The condition had slowly developed after a fall on the back at ten, which caused imperfect paraplegia, motor and sensory. A year later, during pleurisy, he had thrombosis of the left femoral vein. In an excised fragment of the gastrocnemius, the muscular fibres were large, the maximum being $\frac{1}{176}$th of an inch; they presented fatty and vitreous degeneration, and fat was seen between the fasciculi. The muscles of the other leg were somewhat wasted, but the fibres were also enlarged and degenerated. It would appear as though the condition had resulted from a traumatic lesion of the spinal cord, and had been intensified in the left leg by the influence of the thrombosis and resulting vascular disturbance.

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* Auerbach, 'Virchow's Archiv.,' Bd. liii, pp. 234 and 397; Berger, 'Deut. Archiv f. klin. Med.,' Bd. ix, 1872, p. 363; Friedreich, 'Ueber Prog. Muskelatrophie, &c.,' 1873; Eulenberg, 'Real-Encyclopädie,' Bd. ix, p. 354. A very similar case is described by Pal, 'Wien. klin. Wochenschr.,' 1889, No. 10.

† 'Deutsch. Med. Wochenschrift,' 1885, No. 12.

THOMSEN'S DISEASE; MYOTONIA CONGENITA.

The malady thus designated may be considered here, notwithstanding the obscurity of its nature, because it agrees with the diseases last described, in that the symptoms are muscular, and that the disease seems often congenital and occurs in families. It has been named after the physician whose description gained for it general notice, and who is himself its subject, but it had been previously described by Leyden, and hinted at, long ago, by Sir Charles Bell.* It is not always congenital, and "Transient Myotone" would be a more exact name.

The disease is characterised by a peculiar rigidity of the muscles, which comes on when they are first put in action after a period of rest. The rigidity is transient, and when it has passed off, it does not return as long as exertion is continued. The malady is often hereditary, and usually affects several members of the same family. In that of Thomsen, cases can be traced through five generations. It appears to be more common in Scandinavia and Germany than in France or England, but it is a rare disease;[*] and although a considerable number of clinical cases have now been recorded, there has been no post-mortem examination. There is little to attract attention in the slighter forms, and the rarity of these may ultimately be found to be less than at present appears.

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* Bell's 'Nervous System,' Case 184, p. 436. The objections to cognominal nomenclature of disease are certainly reduced to a minimum when the first describer of a rare disease is also its subject. The most important papers on the disease are those of Leyden, 'Klinik der Ruckenmarkskr.,' 1874, Bd. i, p. 128; Thomsen, 'Arch. f. Psychiatrie,' Bd. vi, 1876, p. 702, also 'Centralbl. f. Nervenkr.,' 1885, p. 193; Bernhardt, 'Virchow's Archiv,' Bd. lxxv, 1879, p. 516, and 'Centralblatt f. ervenkr.,' 1885, p. 122; Ballet and Marie, 'Arch. de Neur.,' 1883, No. 13; Möbius, 'Schmidt's Jahrb.,' Bd. cxcviii, 1883; Ringer and Sainsbury, 'Lancet,' 1884, pp. 767, 816, and 860; Erb, 'Die Thomsen'sche Krankheit,' Leipzig, 1886, also 'Deut. Archiv f. klin. Med.,' 1890, xlv, p. 529; Seifert, ib., xlvii; Marie, in 'Encycl. des Sc. Méd.,' 1886; Hale White, 'Brain,' April, 1886, and 'Guy's Hosp. Rep.,' vol. xlvi (a very valuable paper, with bibliography); Jacoby, 'Journ. of Neur. and Ment. Dis.,' 18..7, xiv, p. 23; Jolly, 'Südwest. Neurol.,' Baden, June, 1890; Buzzard, 'Lancet,' 1887.

Both sexes suffer. In most cases the symptoms have been first noted in childhood, between four and ten years of age, sometimes even in the cradle. They increase during the period of muscular development, and then remain stationary. A few patients have seemed free until about the period of puberty, but even in such instances it is probable that the tendency was congenital in origin, since the cases occurred in families, other members of which suffered earlier. But in a few cases symptoms apparently identical with those of the congenital disease have come on in early manhood after some exciting cause—in one case a lightning stroke, in another a sudden alarm[†] Severe and prolonged exertion during two years preceded the onset of very characteristic symptoms in a man of twenty-five, without heredity.[‡] In such cases the disease has been apparently acquired.

The characteristic symptom is tonic spasm of the muscles when they are put in action after a period of rest. As soon as the patient attempts to move, the muscles become rigid. The rigidity may make movement impossible while it lasts, or may merely lessen the possible range of movement. After a few minutes, or less, the spasm passes away, to be renewed, but in slighter degree, by a fresh attempt. If the movements are continued, the spasm soon becomes trifling, and does not return until after a period of rest. The longer the rest, the more troublesome is the spasm. The subject can walk for hours without fatigue when it has passed off. The rigidity is sometimes lessened by alcohol and increased by attention and by fear of it; the more the sufferer tries to overcome the stiffness, the less is he able to do so. It is also worse in cold and damp weather. In a severe case, a slight impulse will make the person fall, and it may then be impossible for him, for some minutes, to rise from the ground. The arms are usually less affected than the legs, but in some cases, the rigidity fixes the fingers for a short time on an attempt to use the hand. The muscles of the face are usually free; mastication may, however, be interfered with by the spasm in the muscles of the jaw. Rarely, the tongue or face has been affected; still more rarely the muscles of the eyeball, interfering with its movement and retarding the descent of the upper lid.[§] Most voluntary muscles, indeed, seem liable to suffer, even the muscles of the larynx and pharynx; those concerned in the processes of respiration, defæcation, micturition, and coitus have also been involved. Sometimes the spasm is greater on one side of the body than on the other.

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[*] In England cases have been observed by Buzzard, Herschell, Hale White, Banham, and Chapman.

[†] 'Engel, 'Phil. Med. Times,' 1883, p. 412; Schönfeld, 'Berlin. med. Wochenschr.,' 1883, No. 27.

[‡] Moyer, 'Med. News,' 1890.

[§] Raymond, 'Gaz. Méd. de Paris,' June, 1891.

The muscles are always well nourished; they are often, indeed, above the normal size, and possess more than normal strength, but they are sometimes less strong than is normal, even when large. Thomsen believes that the more the muscles are employed the less severe is the spasm, and that a life of active exertion produces some permanent amelioration in the disease. The malady is, however, a source of some disability, and of considerable annoyance; in the words of Thomsen, "it casts a shadow over the lives of the sufferers," and may have caused the mental irritability and hypochondriacal tendency that have been conspicuous in some cases.

Careful investigations of the muscular phenomena have been made by several observers and that by Erb is especially instructive. In his case a single brief effort caused tonic contraction which lasted for twenty-five seconds. Momentary electrical stimulation of the nerves caused only a momentary contraction of the muscles, but continued stimulation always causes a prolonged contraction, the "myotonic reaction" of Erb. In certain muscles, moreover, an uninterrupted current caused peculiar wave-like contractions, about one per second, passing from the negative to the positive pole (Erb).[*] Any strong stimulation of the muscle itself caused a prolonged after-contraction.[†] The irritability of the nerves is generally normal in degree; that of the muscles is normal or increased to voltaism. In some cases the anodal closure contraction has occurred with undue readiness, and the "latent interval" after stimulation has been found unchanged (Hale White) or increased.[‡] The muscles are remarkably sensitive to mechanical stimulation, and firm pressure may cause a tonic contraction lasting from twelve to twenty seconds.

In all undoubted cases of this disease sensibility has been intact, and the superficial reflexes have been unaltered. Myotatic irritability is normal or increased.

Once developed, the condition seems to persist, with little change, through life, but in one case, a female (in whose family were other cases) considerable improvement is said to have followed marriage.[§] In some instances, the symptoms are slight, and remain so, even when they begin in early life and occur in the same families as severe cases. Such cases, when isolated, may readily be overlooked.

Pathology.—An examination of the central nervous system has yet to be made, but it is unlikely to yield results of novelty or importance. The state of the muscular fibres has been ascertained from excised fragments—a method which, however, involves the fallacy, already mentioned (p. 534, note), of possibly stimulating the fibres to contraction. At the same time, the evidence of hypertrophy appears conclusive. Where the minimum width was the same as in health, the maximum diameter was double the normal (Erb). In other cases the maximum or even average diameter in fragments excised under chloroform was double that observed after death in unaffected persons; and that the size was pathological was rendered probable by the aspect of the fibres, which presented indistinct striation, fewer "sarcous elements," irregular, non-parallel edges, and sometimes vacuolation. An increase of the nuclei and of the interstitial connective tissue has been found.*

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[*] Although Erb, Seifert, and others have noted this, some observers (as Hale White) have failed to obtain the phenomenon.

[†] Jolly has found that repeated stimulations by either current, without too long an interval between the successive stimulations, tend to render the after-contraction less and less evident, until it ceases to occur ('Neur. Centralbl.,' 1890, p 438).

[‡] ·025 or ·03 sec. instead of ·01 (Bluminau, 'Neur. Cent.,' 1888, p. 679).

[§] Herschell, 'Lancet,' Feb. 1, 1890.

The manifestation of the disease is in a disorder of the functions of the muscles, and most writers on the subject have followed Leyden in regarding the malady as essentially muscular in nature, as consisting in an altered functional condition of the muscular tissue. This opinion is strongly supported by the frequent alteration in electrical excitability (which can hardly be otherwise interpreted than as an indication of a change in the mode of action of the contractile muscular protoplasm), by the structural changes, and also by the interesting experiments of Ringer and Sainsbury, who found that certain salts, such as sodium phosphate, are capable of causing in the frog tonic spasm bearing considerable resemblance to that of Thomsen's disease, and that such spasm persists not only after the nerve has been divided, but after the intra-muscular nerve-endings have been paralysed by curara. This does not prove that Thomsen's disease is muscular in origin; it is a proof that a similar state may be due to the muscular tissue, but no proof that this is the sole element in Thomsen's disease. We must remember that the malady has apparently resulted, in rare cases, from influences acting on the nervous system in adult life, and also that, in the congenital cases, the spasm only develops, as a rule, after some years of voluntary action. It may thus be an acquired disease, and the case related at the end of this chapter shows that similar spasm may be due to a primary affection of the cord. The spasm is a transient consequence of rest. But "rest" is not inactivity, either in the nerve-cells or muscles. Muscular "tone" and adaptation to posture mean an unceasing flow of nerve-force from the cells, an overflow perhaps, due to the perpetual elaboration of energy which keeps the cells ready for instant response to the voluntary stimulus. The tonic activity then ceases, or at least does not interfere with the different action excited by the will. But the phenomena of Thomsen's disease suggest that the cells respond abnormally to the voluntary stimulus, that this causes at first an increased tonic activity, slowly ceasing as their energy is lessened by action. Wide as is the difference between the muscular and nervous tissues, we must remember that they have some conditions in common. The dependence of the nutrition of the muscles on that of the motor nerve-fibres and cells is a very remarkable fact, and so also is the influence of the functional activity of the cells and fibres in causing a similar condition in the muscular tissue. To say that the two structures are connected is hardly an explanation of the fact. Whatever is the nature of the relation between them, it is at least conceivable that an abnormal functional state, congenital in origin, may be common to the two, and that the peculiar over-action in the muscles may be accompanied by a similar over-action in the ganglion-cells of the spinal cord, and even in the pyramidal cells of the cerebral cortex. It is even conceivable that the condition of the nerve-cells may be the primary change, and that of the muscles may be secondary, although, when produced, it is in some degree independent, and may be excited independently by local stimulation. Such a theory enables us to understand the two facts above mentioned —the influence of emotion and the acquisition of the disease, each of which seems to be inconsistent with a purely muscular pathology.

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* Erb, Seifert, and also Nearonow, 'St. Petersburg psych. Gesellsch.,' 1889; see 'Neur. Cent.,' 1889, p. 239. Hale White found no increase in the nuclei.

Treatment.—No treatment, properly so called, appears to exert any influence on the disease. The congenital malady persists through life; in the cases in which a similar condition has apparently been acquired, it has also been persistent. The only influence that has appeared to Thomsen to ameliorate the condition is a life of active muscular exertion. But it is possible that the influence of therapeutics is not yet exhausted.

Congenital Paramyotone.—Under this name Eulenberg has described* a strange family affection, allied to Thomsen's disease in its general character, although differing very much in its special features, and equally obscure in nature. The malady was widely spread in the affected family, and could be traced through six generations, but appears now to be dying out. Its congenital character was shown not only by its multiplicity, but also by the fact that in some individuals it was manifested immediately after birth. The symptom was tonic spasm, lasting from a quarter of an hour to several hours, excited chiefly by cold, although often by merely slight cold. The rigidity was followed for a time by weakness. The facial muscles were very prone to become thus rigid, especially the orbiculares palpebrarum and oris; and while the contraction lasted the patient was often unable to speak or to open the eyes. The rigidity was slighter in the legs than in the arms, but the subsequent weakness was equally marked. Warmth removed the spasm. There was no persistent loss of power, and no increase of mechanical irritability. The electrical excitability of the nerves was normal; that of the muscles was lowered to each current, and there was an abnormal tendency to tetanic contraction during the passage of the current. Eulenberg speculates that the symptoms may be due to reflex vaso-motor spasm in the muscles, because the diminution of the blood-supply to muscles renders them weak; but it is clearly equally possible that the two certain phenomena, the sensory impression and the muscular contraction, may be directly connected.

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* 'Neurologisches Centralblatt,' 1886, p. 265.

Ataxic paramyotone seems the best provisional designation for an acquired condition of which one case has come under my notice. Persistent tonic spasm, like the transient spasm of Thomsen's disease, was associated with distinct ataxy, and also with weakness and some anæsthesia. The symptoms commenced gradually in the legs, in a healthy man, aged 40, without neurotic heredity; it invaded the arms six months later, and increased more rapidly in the arms than in the legs. At 41½ his state was this. A well-built man, with well-developed muscles, unduly firm in all parts except the neck and head. The firmness was due to tonic spasm, which never ceased. It was present on waking in the morning, and through the day interfered with all movements, making them slow and stiff. He rose from a seat slowly and with difficulty; when standing there was slight unsteadiness, increased by closing the eyes, and a slight impulse or stumble in walking would cause a fall, chiefly because the spasm prevented the needed quickness of movement to save him. Prolonged exertion had no influence on the tonic spasm, which opposed passive, as much as active, movement. Power in arms and legs was lessened, but not considerably. The grasp = 50 and 55 ko. The extensors of the fingers were weaker than the flexors, but could act perfectly although involuntary flexion followed the slow extension. The electrical irritability of the muscles seemed to be normal. Inco-ordination was especially marked in the hands; with eyes shut, buttoning his coat was almost impossible; the attempt was made with thumb and forefinger, the others being flexed, and he could touch his nose only after many wandering failures. On the palms, from the wrist to the tips of the fingers, sensibility was lost to touch, slightly delayed to pain, little changed to temperature. Slight tactile loss extended to the back of the last phalanges; on the back of the hands it was normal. A similar but slighter loss existed on the soles; he often felt as if walking on a rounded surface. The size and weight of objects placed in the extended hand could not be recognised. No trace of the knee-jerk or other evidence of myotatic irritability could be elicited, but the spasm was enough to explain the failure, and its real condition is uncertain. Mind, special senses, and cranial nerves were unaffected.

Unfortunately, the course of the affection could not be observed. Its symptoms must have been due to disease of the spinal cord, and they are instructive as showing that muscular spasm, like that of Thomsen's disease, may be thus produced. The time may not have been sufficient for the production of changes in the irritability of the muscles, but we must also remember that differences in the influence on the muscles may be connected with the defective power.

TUMOURS OF THE SPINAL CORD.

Morbid growths within the spinal canal may spring from the membranes, or may grow in the substance of the cord itself. The difference in seat entails some difference in symptoms, but it is nevertheless convenient to consider the two classes together, because they have many symptoms in common, and it is often impossible to carry the diagnosis farther than the existence of an intra-spinal tumour.

Etiology.—The general causes of these growths correspond, for the most part, to those concerned in their production in other situations, and present few peculiarities that merit special mention. Fatty growth outside the dura mater occurs early in life; malignant tumours develop late. Of tumours within the dural sheath, myxomata are chiefly met with in middle life, tubercular growths occur occasionally in childhood, but generally between fifteen and thirty-five (Herter*); lipomata are congenital. Males are a little more prone to suffer than females.†

Of the diathetic conditions which give rise to tumours elsewhere, only two, syphilis and tubercle, are effective in causing growths which commence within the spinal canal. Parasitic tumours occur, due to the same influences which produce them in other situations. A few rare growths appear to be congenital in origin, due to the abnormal development of germinal tissue, while the more common gliomata within the cord arise from embryonal tissue, which has suffered an arrest of its normal development. Of the causes of other forms of tumour we know practically nothing. Injuries, such as a blow on the spine, have been supposed to be occasional causes, and their influence in rare instances has seemed possible, but the evidence is not so strong as it is in the case of tumours in some other situations. In many cases the first symptoms have immediately followed some exposure to cold and wet, and it seems probable that this influence may have excited secondary processes in the nerve-elements which were already deranged by the growth, and may thus have frequently excited the first symptoms of the tumour, but it can have had no share in the production of the growth itself. Traumatic hæmorrhages may, however, result in cystic formations, which have been mistaken for new growths.

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* Herter, 'Journ. of Mental Dis.,' 1890, has analysed twenty-six cases.

† A collection of facts relating to these points, by Mr. Victor Horsley, will be found in the paper written by him and myself on the case of successful removal of a tumour from the spinal cord, 'Med.-Chir. Trans.,' 1889; but the numerical basis is far too meagre to supply more than statistical suggestions. It will be necessary to wait for a considerable time before adequate data are obtained.

PATHOLOGICAL ANATOMY.—The growths within the spinal canal may develop outside the dura mater, inside the dura mater, or within the substance of the cord. The extra-dural tumours may spring from the membrane, or from the tissue between the membrane and the bone, or may grow into the canal from the outside, through the intervertebral foramina. Subdural tumours may proceed from the inner surface of this membrane, from the arachnoid, or from the pia mater (Fig. 165). The growths in the cord may spring from the pia mater, or may develop in the substance of the cord. They sometimes proceed from the peculiar tissue which surrounds the central canal.

The forms of extra-dural tumours are lipoma, from an overgrowth of the fat which normally exists between the membranes and the bone; and parasitic tumours, chiefly echinococci; but all are rare.* Growths also occur that spring from the bones or intervertebral tissue—enchondroma, sarcoma, and cancerous tumours, which have been already considered. Far more frequent are collections of inflammatory products from bone disease, but these do not come into the category of morbid growths.

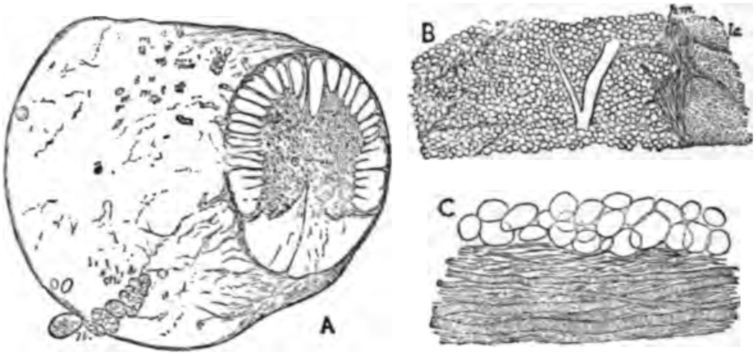

FIG. 163.—Myolipoma of the spinal cord. A, transverse section of the tumour and of the conus medullaris of the spinal cord, to which the growth was attached, and of which the grey matter is represented by the dotted shading O M. *n*, sections of nerve-roots, partly enclosed in the tumour; *m*, bundles of muscular fibres. B, part of the tumour more highly magnified, showing the fat-cells, of which it was chiefly composed; *p.m.* pia mater of the cord, of which *l.c.* is part of the lateral column. C, part of the tumour, still more magnified, showing striated muscular fibres, fibrous tissue, and fat-cells (see also Fig. 157, p. 517). The tumour had caused no symptoms.

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* An adeno-sarcoma has also been met with (Hodenpyl, 'Am. Journ. Med. Sc.,' 1888).

The tumours which begin within the dura-matral sheath are chiefly syphilomata, sarcomata, and myxomata, sometimes containing cysts or "brain-sand." Tubercular and parasitic tumours are rare, but both echinococci and cysticerci have been met with, developing in the meshes of the arachnoid. Fatty tumours have been found in a few instances, and may contain muscular fibres. They are congenital and sometimes associated with spina bifida (q.v.). Fig. 163 represents such a tumour, a myolipoma consisting of fatty tissue and striated muscular fibres, which had grown from the pia mater or meshes of the arachnoid, and was found in a case of tabes.* Neuromata occur on the nerve-roots, and may compress the cord.†

The growths within the substance of the cord itself are more diverse in character. Syphiloma and glioma are the most common; sarcomata, myxomata, and tubercular tumours also occur, containing the characteristic bacilli, and always associated with tubercle elsewhere. A cysticercus has once been met with. Some growths have a compound character, and have been termed myxo-sarcoma, glio-sarcoma, and fibro-sarcoma. Sarcomata and gliomata are sometimes very vascular, and have been termed "angio-sarcoma" and "angio-glioma." It is far more common for the tumour to spring from the pia mater or from the peri-ependymal tissue around the canal than for it to begin among the nerve-structures, but tubercular growths usually begin in the grey matter on one side.

Extra-dural growths are always single; those within the dura mater are often single, but sometimes two or three co-exist; or the growth may be diffuse, as in a sarcoma of the pia mater, which extended as high as the sixth pair of cerebral nerves, and as low as the cauda equina.‡ Neuromata or sarcomata on the nerve-roots are often multiple, and sometimes are very numerous. Tumours within the spinal cord are also usually single, even those of tubercular nature, but occasionally more than one growth is found.

The size attained by growths outside the cord is necessarily moderate, in consequence of the limitation of the space in which they grow. They vary from the size of a pea to a width of one and a length of two inches, which is rarely exceeded, and chiefly by extra-dural tumours. Those that develop from the pia mater are usually smaller than those that spring from the dura mater. Multiple tumours on the nerve-roots are generally small. Adhesions often form between the growth and the membrane from which it does not arise. The tumour compresses the nerve-roots and the spinal cord (Figs. 164, 167). The compression produced by tumours within the dura mater is always greater than that by tumours outside this membrane.

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* See 'Path. Soc. Trans.,' vol. xxvii, 19. Another rare case is one of lymph-angeioma of the pia mater (Traube, 'Neur. Cent.,' 1887).

† Myxo-fibromata may also form on the nerve-roots, and even extend on to the cord. See Bruce and Mott, 'Brain,' July, 1887.

‡ Pasteur, 'Brit. Med. Journ.,' July, 1887.

FIG. 164.

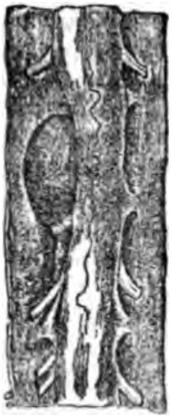

FIG. 165.

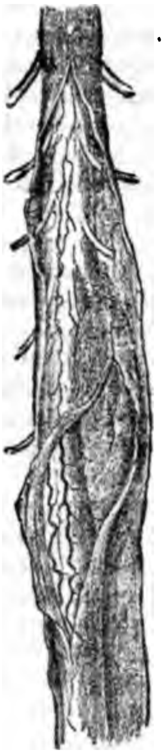

FIG. 166.

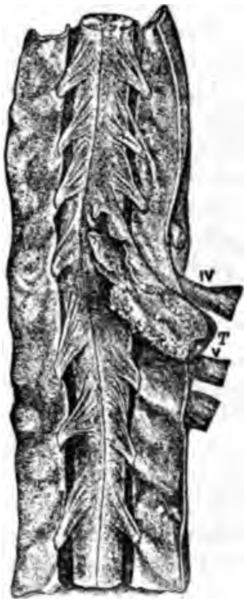

FIG. 164.—Tumour growing from the inner surface of the dura mater, and compressing the spinal cord in the mid-dorsal region. The tumour, 3 cm. long, was a sarcoma in which the cells were arranged in concentric groups, the centres of which had undergone calcification. The spinal cord was softened and dark in tint at the compressed part.* (After Lancereaux.)

FIG. 165.—Sarcoma growing between the arachnoid and pia mater in the mid-dorsal region of the cord. The tumour had sprung from the meshes of the arachnoid, and was only slightly adherent to the pia mater. The spinal cord was compressed and softened. In the figure the dura-matral sheath has been laid open, except at the top, and the arachnoid has been opened over the lower half of the tumour, which, 5 cm. long, lies on the posterior and (right) lateral aspect of the cord.† (After Lancereaux.)

FIG. 166.—A tumour (T) growing from the dura mater, and compressing the right side of the spinal cord at the origin of the 4th, 5th, and 6th cervical nerves. The tumour was a spindle-celled sarcoma springing from the arachnoid.‡ (After Leyden.)

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* The patient was a woman seventy-one years of age, paraplegic, with strong flexor contracture of the legs, loss of power over the sphincters, and considerable impairment, but not absolute loss, of sensibility. The symptoms came on gradually six years before death. (Lancereaux, 'Atlas d'Anat. Path.,' p. 444.)

† The patient was a woman twenty-eight years of age. The first symptom was pain, radiating over the upper part of the abdomen on the right side, and afterwards extending to the leg and to the left side. After a few months, weakness of the legs gradually came on, and became absolute, with loss of sensation and of power over the sphincters. Extensive bedsores were the immediate cause of death, which occurred eighteen months after the onset. (Lancereaux, 'Atlas,' p. 447.)

‡ The patient was a man aged thirty-five. At twenty-eight pain commenced in

Fig. 167.

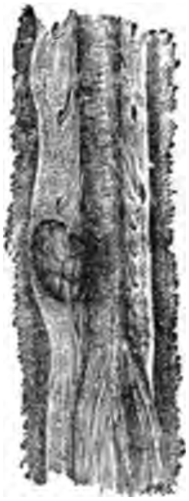


Fig. 168.

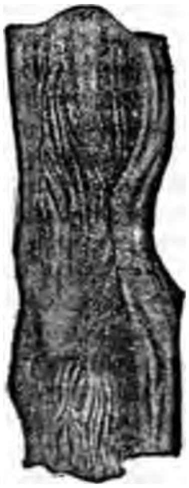


Fig. 169.

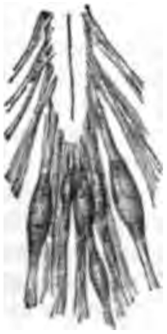


Fig. 167.—Tumour of dura mater opposite the upper part of the lumbar enlargement, compressing nerve-roots and spinal cord. (From a preparation in University College Museum. Drawn by Dr. H. R. Spencer.)

Fig. 168.—Tumour of the cauda equina surrounding and enclosing many of the nerve-roots. The cord itself was unaffected. The structure of the tumour was that of a fibro-sarcoma.*

Fig. 169.—Neuromata of the cauda equina. (After Lancereaux.) The growths had apparently given rise to no symptoms.

the right forearm, and continued, intermitting, for some years, until, at thirty-four, it extended through the whole arm to the neck beside the spine, where it was increased by movement. Similar pain afterwards came on in the left arm. Then followed weakness in the right arm, slight spasm in the right leg, and tingling in the left. The arm became almost powerless and wasted, the right leg weak, and flexion of the neck caused severe local pain. These symptoms continued and increased, and pain in the left leg came on. The cervical spine became tender, and movement of the head to the right was limited. Then both legs became weak, and sensibility was lost as high as the nipples. The paraplegia became absolute, bed-sores formed, and the patient died seven years after the onset of the symptoms. Softening of the spinal cord extended down into the dorsal region. (Leyden, 'Klin. der Rückenmarkskr.,' Bd. i, p. 450.)

* The patient was a man aged twenty-eight, whose symptoms commenced nine months before death, and consisted in severe pains in the legs and progressive weakness, the power of standing being lost about four months after the onset. A little power in the flexors of the hips and the extensors of the knees persisted almost to the last, but the knee-jerk was lost. All the muscles of the legs wasted, those below the knee extremely, and even when the patient was first seen, electrical irritability was greatly lowered to both currents, without any reaction of degeneration. Tactile sensibility was impaired in each foot and lower leg, more in the right than in the left, and chiefly in the region supplied from the sacral plexus. Sensibility to pain was not affected. The bladder was affected early, and symptoms of pyelo-nephritis already existed. The kidney disease was the immediate cause of death, which was preceded by several convulsions, apparently uræmic. A microscopical examination showed considerable damage to many nerve-roots involved in the tumour, while others had escaped. The muscles presented intense granular degeneration with some longitudinal striation, and increase of the interstitial nuclei.

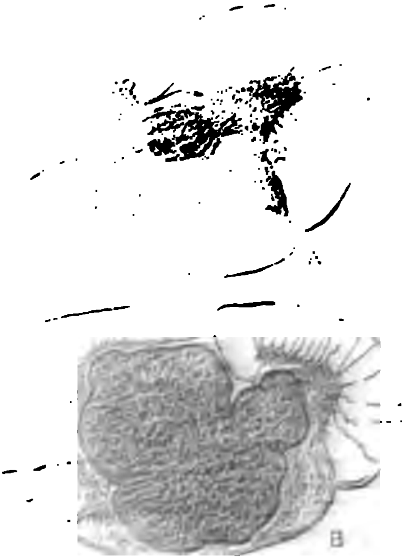
The amount of pressure is proportioned to the size of the tumour and its consistence. A soft growth outside the sheath may attain a considerable size, and even extend through the intervertebral foramina, without exerting much pressure on the cord itself. At the compressed part the cord is narrowed and softened, usually indented or flattened, because the pressure is either from one side, from the front, or from the back. Rarely, tho degree of pressure is so great that the cord is reduced to the size of a crowquill, and it has even been apparently interrupted at the spot, the upper and lower portions being conical, and their pointed extremities connected only by membranous tissue. The softening of the cord depends on inflammation, the "pressure-myelitis" considered in a preceding chapter (p. 345), and it presents the tissue-changes that have been there described. Secondary inflammation may extend down the cord below the growth, or for a short distance above it, often unequally in the different elements —sometimes in the grey matter into the lumbar enlargement, and even on one side only (Francotte, see p. 552). The usual secondary degenerations are commonly conspicuous.

The growths that involve the cauda equina often attain a larger size than those that occur higher up, because this part of the vertebral canal is large, and the nerve-roots occupy but a small part of it. Most tumours in this situation are sarcomata or fibro-sarcomata, but it is a not uncommon seat of syphilitic growths. They usually spring from the tissue of the arachnoid, and often surround and include the nerves (Fig. 168), the amount of damage to which is very variable.

Multiple tumours outside the cord are sometimes very numerous. They are usually sarcomata, and spring from the membranes and sheaths of the nerve-roots. They are various in size, ranging from that of a hazel nut to that of a pin's head, and many very small growths are often scattered among the nerves of the cauda equina. In some cases of this character similar growths have existed in the cerebral membranes.

The tumours within the spinal cord are usually small, seldom exceeding half an inch in diameter, and usually less, even when they give rise to considerable impairment of function. But their vertical extent often exceeds their transverse diameter, and sometimes they grow through a considerable extent of the spinal cord. Gliomata, and especially the gliomatous growths that spring from the central region, and arise from embryonal neuroglial tissue (persistent by arrest of development, and often associated with syringo-myelia), are those which have most frequently a considerable vertical extent. Such a glioma has been known to reach from the medulla oblongata to the lumbar region, and often extends up from the cervical region to the base of the brain.

The cord is enlarged at the seat of a tumour, and the precise character of the enlargement depends on the position of the growth. In an infiltrating glioma of the upper part of the cord, the increase [illegible] the foramen magnum [illegible] the abnormal [illegible] the surface. The consistence [illegible] soft growths, but the tumour [illegible] normal cord, and the difference is [illegible] the vicinity of the growth. If [illegible] the pia mater and arachnoid may be [illegible] may even be adherent to the dura [illegible] extension of the meningitis to other [illegible], is usually very distinct, since its [illegible] the nerve-substance. Only glioma and [illegible]oma resemble the normal [illegible] substance in appearance, [illegible] the new tissue is usually distinct in position. These tumours in some cases blend with the substance of the cord, but in other instances they are bounded by an area of softening which often isolates even invading growths. Some tumours are sharply limited. It may be difficult to say in what structure the tumour began, but this may sometimes be ascertained by an examination of the upper and lower parts, where the growth is commonly limited to that part of the cord in which it first originated. Thus in Fig. 170, B, the tumour occupies a very large area, although the nearly equal extent on each side suggests its central origin, and this is clearly seen in the section through the upper part of the growth, in which the posterior commissure can be traced in front and behind the small area of growth, pointing to the peri-ependymal tissue as its source. Syphilitic tumours commonly originate in the pia mater, as they do also in the brain, and the membrane in their vicinity is often thickened. They both invade and [illegible] the nerve-tissue. An instance of such a gumma of the cord



[illegible] of spinal cord, springing [illegible] around the central canal [illegible] the tumour in the middle of [illegible] the growth is here within [illegible] section through [illegible] of the tumour which occupies [illegible] of the cord. The [illegible] was a sarcoma [illegible] spindle-cells arranged [illegible] concentric layers. The [illegible] suffering [illegible] from a cerebral [illegible] hemiplegia, and the spinal [illegible] apparently developed rapidly [illegible] and prostration [illegible] not suspected during life.

is shown in Fig. 171. It probably commenced at the furrow opposite the head of the posterior cornu, which it has destroyed, and the pressure which it exerted is evident from the displacement of the posterior median septum. In all forms of tumour the central canal is often obliterated by the pressure, and may be slightly dilated higher up; this dilatation is especially common from growths that begin in the peri-ependymal tissue. The considerable enlargement of the canal, termed "syringo-myelia," is generally associated with a peculiar growth in this position, probably congenital in origin; the condition is described in another chapter. In other forms of tumour, cysts occasionally develop, and hæmorrhage may occur into such cysts, or into the softened tissue in the vicinity of the tumour, or even in the substance of a soft growth itself, especially when this is a glioma. From the region of the growth, secondary degenerations can frequently be traced upwards and downwards, but these are often slighter than the apparent damage would suggest, sometimes by reason of the remarkable tolerance of the nerve-fibres to pressure if this is slowly developed, sometimes because a rapidly growing tumour has not given time for them to become conspicuous. As regards vertical position, growths

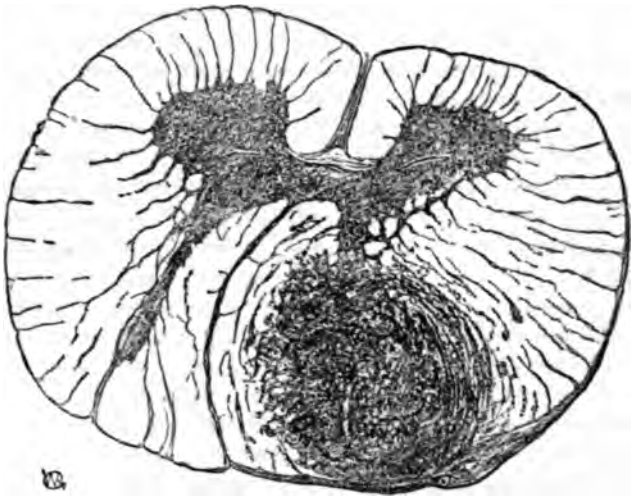


FIG. 171.—Syphiloma in the spinal cord, occupying the position of the posterior cornu and adjacent parts of the posterior and lateral columns, in the lower cervical region. The tumour presented the usual combination of grey translucent, and yellow cheesy areas. A second similar, but much smaller tumour existed higher up. The symptoms were complicated by hemiplegia, &c., of cerebral origin, due to a gumma in the brain. Paralysis with rigid flexor contracture of the left arm and leg was apparently due to the growth. The disease was certainly syphilitic, but it is noteworthy that it had developed during continuous treatment by full doses of iodide, which had entirely removed earlier symptoms, but to which the system had apparently become accustomed.

may occur in any part, but are said to be most frequent beneath the lower cervical spines, and in the upper and lower dorsal regions, because (it is conjectured) there is least room at these places, although why the result should follow from the cause is not clear.

SYMPTOMS.—The symptoms produced by tumours within the spinal canal, whether these are within the cord or outside it, resemble in their general characters those which we have already considered as resulting from growth and caries in the bones of the vertebral column. The resemblance is most close in the case of extra-dural growths, and least so in that of tumours within the substance of the cord. The chief differences are the relative preponderance of symptoms of early irritation of the cord itself when the growth is within it, and the frequent indications of a transverse extension of the damage from one side of the cord to the other.

In the majority of cases pain is prominent through the whole course of the disease. It is usually the earliest effect, severe both before and after the development of other symptoms. The pain may be intense along the course of the nerves which arise at the level of the tumour, and also in the parts supplied by nerves below that level, *e.g.* in the legs, but it is not felt above the growth. It may be a sharp acute pain, but has often a "burning" character, and is sometimes "stabbing" or "rending." Dull aching pain may be felt between the attacks of severe pain, or before them, and may then give rise to the most frequent of all diagnostic errors—mistaking grave organic disease for simple rheumatism, on account of the similarity in the character of the pains. The intensity of the severe pain is usually very great, and it has more than once led the unhappy sufferer to attempt suicide. The pains are often felt on one side first, in one arm, in one side of the trunk at a given level, or in one leg, and a considerable time may elapse before they extend to the other side. Occasionally the pain is bilateral from the first. It is generally greatest when the tumour compresses the cord from behind forwards, or *vice versâ* (Horsley); this is open to more than one explanation. Pain is sometimes increased by movement, but rarely in the intense degree common in tumours of the bone. When the growth is in the lower lumbar region, the pain is often first referred to the soles, and seems to ascend. The sensory loss due to subsequent nerve destruction, follows a similar course. Pain is sometimes felt in the spine, frequently when the growth begins in the dura mater, rarely when it is within the substance of the cord. Tenderness of the spine often exists in the former cases, but is on the whole not common. Other subjective sensations may occur with the pain or in the intervals—"numbness," tingling, formication, &c., and they give significance to the pains as an indication of organic damage to the nerve-structures. The root-pains in the trunk are often accompanied by a sense of constriction, which may be very distressing. Hyperæsthesia of the skin frequently accompanies pain felt at the level of the tumour, less commonly those in the parts below. Very rarely pain is absent; chiefly in extra-dural lipoma, probably from the nature of the growth.

Muscular spasm is another common symptom, most pronounced when the tumour springs from the membranes. There may be some rigidity of the back opposite the seat of the growth, usually associated with local pain. It is most marked when the disease is at the more mobile parts of the spine, especially when it is in the cervical region. In such cases the rigidity may be painful, and the pain may be increased by movement. Severe spasm in the abdominal muscles is often associated with severe girdle-pain. Contractures may develop in the limbs, both in those supplied by the nerves which arise at the level of the tumour, and are directly irritated by it, and also, although less commonly, in the parts supplied from the spinal cord below the level of the growth. Thus the tumour in one half of the cord in the cervical region (shown in Fig. 171) caused persistent flexor contracture in the arm and leg on the side of the growth, and in the early stage of dorsal tumours one leg only may be rigid, or more so than the other. The seat of rigidity thus needs to be specially noted, as well as its character, if we are to obtain from it diagnostic guidance, because it may be due to the irritation, either of the nerve-roots or of the conducting tracts. Both forms sometimes occur before loss of power, but pain generally precedes them.

Paralysis, of gradual onset, is almost invariably one of the symptoms. Paraplegia is the most common form, but all four limbs may be paralysed by a growth in the cervical region. Very commonly one leg becomes weakened before the other, and occasionally one arm and leg suffer before those of the other side. Tumours which are situated in the middle line or in the centre of the cord, however, usually affect both sides at the same time, and the effects of a congenital growth may thus accompany and complicate those of an associated syringomyelia (*q. v.*). The proximal parts of the limbs seem, as a rule, to be first affected, although exceptions are occasionally met with.

The palsy, gradual in onset, is usually also slow in its extension, the rate of its increase depending on the rate at which the tumour grows and compresses the spinal cord. This is the characteristic course of the paralysis produced by a tumour. But in a large number of cases the loss of power comes on in a subacute, or even an acute, manner, in consequence of the myelitis excited by the compression, which, in such cases, may run a course of independent rapidity. When the inflammation is thus in excess of its cause, the palsy may lessen as damage from the inflammation subsides to the degree of the damage from pressure. The symptoms that depend on compression slowly but surely increase as the tumour grows, and usually attain a high degree of intensity; the loss of power in the legs, for instance, becoming absolute and remaining so.

Loss of sensation may come on with the loss of motor power; more often after this has become considerable. It may be slight or partial at first, occasionally for a long time, but tends to become complete as the damage to the cord increases, and it often ascends the legs. It corresponds in distribution to the motor palsy, when the disease is below the middle of the dorsal region; but if situated higher, and on one side, the sensory loss is often greater on the side opposite to the motor paralysis. Tumours of the cord furnish a considerable proportion of the cases in which the crossed symptoms of a unilateral lesion are well marked (see p. 229). In addition to the loss of sensation which results from the damage to the cord itself, areas of anæsthesia may exist in the region supplied by the nerves which are damaged by the growth, and in which the severe radiating pains are felt. If there is crossed sensory and motor palsy, these root symptoms exist, chiefly on the side opposite to the anæsthesia in the limbs. Inco-ordination of movement is not a common symptom of spinal tumours, but has been occasionally met with in cases of growth invading the posterior columns, and also as a result of multiple tumours of the nerve-roots (see p. 428). Loss of power usually coexists and quickly becomes predominant when the growth is in the cord.

The condition of reflex action depends on the position of the tumour. In the lumbar enlargement or the cauda equina, a growth abolishes reflex action in the legs, and in any position the trunk-reflexes are lost at the level of the lesion. But when the disease is situated higher up, reflex action is increased, and the excess of cutaneous reflex action is a very marked and early symptom of the disease; it is lessened and lost only when the lower part of the cord is damaged by secondary myelitis (superadded to descending degeneration and distinct from it). The myotatic irritability presents also the increase that invariably results from damage to the pyramidal tracts, and the usual tonic spasm also gradually develops and often reaches a high degree. Its loss, with muscular relaxation, sometimes met with under these conditions, depends on a descending myelitis, sometimes slight and partial.* Muscular contracture is superadded more frequently in tumours than in many other diseases, and rigid spasm in the flexors of the knee and hip may keep the heels in contact with the nates.

Wasting of the muscles is for the most part confined to the regions supplied by the damaged nerve-roots. In tumours of the lumbar enlargement and cauda equina, the atrophy of the muscles of the legs is a very conspicuous symptom. Vaso-motor disturbance sometimes occurs in the early stage of the disease, chiefly in the distribution of irritated roots, where flushing of the skin has been noted, and the so-called "meningitic streak" ("*tache spinale*") may often be produced.

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* As was found to be the case by Francotte ('Fibrome de la Dure-mère spinale,' Liège, 1888) in a case of tumour compressing the upper dorsal region. Slight descending inflammation of the right anterior cornu coincided with flaccid palsy of the right leg. It should have been mentioned that the connection of a similar loss of myotatic irritability with descending inflammation in dorsal myelitis (referred to at pp. 229 and 316) has been proved in two cases by Francotte (loc. cit., p. 9).

Occasionally vaso-motor œdema is a conspicuous early symptom. In the later stages, bedsores are very common and often severe. Dilatation or contraction of the pupil is common when a tumour is situated in the lower cervical region. Loss of control over the sphincters usually accompanies motor palsy in the legs; their state corresponds with that of reflex action (p. 224).

The course of the symptoms is variable, and depends on the rapidity of growth of the tumour, and on the share which secondary myelitis takes in the production of the symptoms. As a rule, the root symptoms occur first, and in growths outside the cord they may exist alone for months, and even, in the case of a slowly growing tumour, for years, before the symptoms of damage to the cord itself are added. On the other hand, they may be entirely absent in growths which commence within the cord itself. In some cases the symptoms are steadily progressive from first to last. In others the progress is intermittent; periods of increase in the symptoms alternate with stationary periods. Their course is variable, even in cases in which the seat of the tumour and its nature are the same, because they depend on the secondary changes in the nerve-elements, and the course of these (such as the myelitis induced by pressure) is subject to variations, the cause of which is often indistinct. Thus the chronic course of the symptoms is often varied by periods of rapid and even sudden increase, especially in the case of growths that involve the spinal cord itself; and symptoms that have thus developed with rapidity are apt (as already mentioned) to subside, if not completely, at least in considerable degree. Hence a rapid increase of the cord symptoms may be followed by positive improvement for a time, but scarcely ever by an actual disappearance of the manifestations of the disease. The most important element in the course of the disease is the tendency to a lateral extension of the symptoms, the expression of the transverse extension of the damage, as the several structures of the cord, at the same level, successively suffer. Although there is sometimes an extension downwards of the interference with the central functions of the cord, when secondary myelitis descends to the lumbar enlargement, there is scarcely ever an extension upwards above the level of the initial interference with function. A small, slowly growing tumour, even within the cord, sometimes causes no symptoms.

The chief differences between the symptoms of tumours of the cord and membranes, besides those mentioned on p. 550, are that, in the latter, the root symptoms are more often severe, and that the cord symptoms occur later, and are at first more limited in extent. Spinal pain, tenderness, and rigidity are more common. In cord tumours the radiating pains, due to irritation of the nerve-roots, are more often trifling or absent, unless the growth begins in the neighbourhood of the posterior horn; then they may be pronounced. The cord symptoms are often bilateral from the first, and in central tumours they may develop equally on the two sides. If they are unilateral at the onset they extend to the other side sooner than in the case of meningeal growths. In the case of tubercular growths the symptoms of irritation are usually subordinate to those of impaired conduction, and pain is felt chiefly in the legs (Herter). Extensive muscular atrophy is more common in tumours of the cord than in those of the membranes (except in the case of tumours of the cauda equina), because central tumours may damage the grey matter through a considerable vertical extent, and are most common in the enlargements, and also because myelitis spreads in the grey matter far more frequently when the exciting growth is in the cord itself, than when it is in the membranes.

In tumours of the cervical region, the interference with movement of the neck, from the rigidity of the muscles, is often well marked, especially when the tumour is in the membranes. The pains due to irritation of the nerves are felt in the arms, and are often associated with muscular atrophy. The excess of reflex action in the legs is very great, and if the influence of the tumour is chiefly on one side, hemiplegic weakness, with crossed anæsthesia, is present.

Growths in the dorsal region of the cord usually cause severe radiating pains in the trunk, often accompanied by an intensely painful sense of constriction, by hyperæsthesia, and sometimes by anæsthesia. The simple, merely sloping course of the dorsal nerves reveals the precise position of the disease more clearly than do the symptoms in the arms when the tumour is in the cervical region. The interference with the trunk-reflexes often corroborates the indications afforded by the subjective symptoms. Spinal tenderness may be also present, but rigidity of the spinal muscles is not common. Reflex action in the legs presents the same excess that results from tumours in the cervical region, but sometimes, especially when the growth is near the lumbar enlargement, or the pressure-myelitis has been acute, the descending myelitis, as we have seen, may abolish the reflex action and remove spasm, if this previously existed.

The symptoms produced by tumours which involve the lumbar enlargement, vary considerably according to the precise position of the growth. The characteristic pains are felt in the legs, and the muscles supplied from the part invaded or compressed undergo atrophy, and present loss of reflex action and of myotatic irritability; but the anæsthesia is often limited in area. A growth in the upper part of the enlargement may cause wasting only in the flexors of the hip and extensors of the knee, with loss of the knee-jerk and anæsthesia in the front of the thigh, while the muscles below the knee present simple palsy with myotatic excess. Thus a man, who had had syphilis, complained of pain in the left groin and thigh, and presented wasting of the extensors of the knee, with anæsthesia in the front of the thigh; there was loss of the left knee-jerk, and a foot-clonus on each side. He recovered partially, but there was doubtless a small syphiloma at the upper part of the lumbar enlargement on the left side, interrupting the reflex arc, damaging the left motor and sensory roots, and compressing both pyramidal tracts. A growth in the membranes adjacent to the middle or lower part of the lumbar enlargement usually causes symptoms of wide distribution, affecting the whole of both legs, and the early symptoms of irritation often involve the distribution of the upper lumbar nerves. But a growth at the same level within the cord may affect chiefly the muscles of the lower leg, and cause anæsthesia in the region supplied from the sacral plexus. There may also be palsy and wasting of all the muscles of both legs, from extensive secondary myelitis, but even then the sensory loss is usually limited. In lumbar tumours there is early palsy of the bladder and sphincters. Examination of the sphincter ani shows absolute paralysis, and not the pure reflex action present when the disease, arresting voluntary influences, is higher up the cord. The tendency to bedsores is commonly strong in tumours of this part.

Growths situated in the cauda equina, which are below, and do not affect, the cord itself, produce symptoms very similar to those due to tumours of the lumbar enlargement, but the symptoms are bilateral from the first in the majority of cases. Rarely one leg is affected alone at the outset. The symptoms usually begin in the lowest parts of the legs, which are throughout involved in greater degree than the upper parts, and the flexors of the hip may escape altogether. Muscular contraction is much less common than in tumours situated higher up. The anæsthesia is chiefly in the region supplied by the sacral plexus; the pains, usually very severe, are first felt in the same region, and there may be severe pain on each side of the sacrum radiating to the region of the sciatic nerves. The muscles below the knee rapidly waste, and often (but not always) present the degenerative reaction. These symptoms were well marked and characteristic in the case figured and described at p. 546 (Fig. 168), and the condition of the muscles suggests that the absence of any voltaic irritability in the muscular fibres was due to their early and intense fatty degeneration. In many cases the symptoms are irregular in distribution, the nerves are included in the growth, and are often damaged unequally, sometimes less than might be expected from the size of the tumour, which may be considerable before its expansion is arrested by contact with the bony walls of the canal. Hence the loss of sensation may be partial, and reflex action may not be completely abolished. In the case above mentioned, for instance, a prick on the sole caused a reflex contraction of the muscles in the upper parts of the legs, but of these alone. In many cases, however, the loss of sensation has been absolute, and reflex action also has been entirely lost. The tendency to trophic changes is great, and an affection of the sphincters is almost invariable. Cystitis and its consequences readily occur, and may reach a high degree, in consequence of residual accumulation, when the patient is conscious only of slight difficulty of micturition.

Multiple tumours may give rise to very complex symptoms. If there are only two tumours, they may be indicated by the successive development of focal symptoms, similar in character but different in seat. In some cases in which there are many growths, only one or two of the largest have caused symptoms, and the smaller tumours have run a latent course. In other cases in which many tumours have caused slight symptoms, these have simulated closely a system-disease of the cord. Thus, in the remarkable case recorded by Hughes Bennett, multiple growths on the posterior nerve-roots produced the characteristic manifestations of locomotor ataxy.* When cerebral tumours have coexisted with multiple spinal growths, the symptoms of the latter have usually been lost in those of the intra-cranial disease, which has often preceded the spinal lesion.

The duration of the symptoms of tumours of the cord and membranes, those of syphilitic nature being excluded, varies from three months to five, or even ten years. Equally variable, but shorter, is the time that elapses before the symptoms reach their full development, which depends partly on the secondary inflammation, partly on the rate of growth of the tumour. From the former cause, the time may be only a few days, when the myelitis is acute; from the latter it may be as short as three or four weeks, in tumours of tubercular or syphilitic nature. In the majority of cases, death ensues at the end of from one to three years from the commencement of the symptoms, but these last longer when the growth is outside the dura mater than when inside the sheath. The immediate cause of death is usually the direct or indirect effect of the tumour; but in syphilis death is sometimes due to disease elsewhere, and in tubercle, frequently to general pulmonary tuberculosis (which usually precedes the spinal growth), or sometimes to meningitis.

DIAGNOSIS.—The evidence of a growth consists in the symptoms of a focal lesion of slow development, and especially in the presence of signs of compression and irritation (pain and rigidity), while the significance of these is increased by indications that the disease began at the surface of the cord, and sometimes by the history of a cause, as syphilis. Although no symptom is actually pathognomonic, yet the combination of the symptoms, and their course, are in most cases sufficiently characteristic. Those of greatest diagnostic importance are, first, the pain that is felt in the parts at the level of the tumour, or below it, and sometimes in the spine; secondly, the progressive paralysis, motor and sensory. Of less but still considerable importance are the rigidity of the muscles of the spine, the muscular contractions in the limbs, and the early and marked excess of reflex action when the cord itself is involved. In the course of the disease the most important features are the steady progress of the symptoms, and especially their commencement on one side and extension to the other. Of causal indications that occasionally assist the diagnosis, the most important are a history of constitutional syphilis, the presence of tubercular disease elsewhere, and, in rare instances, the existence in other parts of such multiple growths as are known to occur also within the spinal canal, especially multiple sarcomata and neuromata.

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* 'Clin. Trans.' 1885.

The diagnosis of multiple growths depends on the recognition of the successive development of the characteristic symptoms in more than one focus. It is rare for more than two of many tumours to cause definite symptoms. The greatest absolute difficulty in the diagnosis of a spinal tumour is presented by the cases sometimes met with, in which an intra-cranial tumour precedes the growth within the vertebral column, and the symptoms of the latter are overlooked in the presence of the severe manifestations of the former. In most cases, however, the additional symptoms, which usually exist, may be detected by careful observation, and their significance recognised. Growths involving the cauda equina cause symptoms like those of tumours in the lumbar enlargement, save that the effects are from the first bilateral in even more pronounced degree, and the evidence of interference with the nerves (loss of reflex action, nerve pain, &c.) is even more conspicuous.

The differential diagnosis involves the distinction from diseases of the vertebral bones, of the membranes, and of the cord itself. Caries of the spine has many symptoms in common with tumour, but the root-pains are rarely very severe, and the effects of compression of the cord are usually bilateral, either from the first or very soon after their commencement; the paralysis seldom reaches a considerable degree in one leg before the other suffers. The signs of bone disease are rarely long absent, and repeated examination usually reveals tenderness and irregularity of the vertebral spines. From growths in the bones of the spinal column, the diagnosis may at first be impossible. But the pain of tumour is rarely increased by movement to the same intense degree as when the growth commences in the bone, unless the tumour is in the cervical region of the cord, and then the greater accessibility of the vertebral column renders the exclusion of bone growths comparatively easy by local examination. In the dorsal region a growth in the bone may long escape detection, but even here the influence of movement on the pain is often extreme, far greater than in the case of tumours within the canal. The effect of the movement of the vertebræ on the nerve-roots, when compressed as they pass through the foramina, is far greater than when they are compressed within the canal. Sooner or later a local enlargement may present itself externally, and show the nature of the case.

" Hypertrophic pachymeningitis," affecting the cervical region, is the only meningeal disease which closely simulates the symptoms of tumour, but its effects are usually from the first bilateral, and they have a considerable vertical extent. Hence it is only with central tumours, which grow through several inches or more of the cord, that the disease is likely to be confounded. In each malady there may be muscular atrophy in the arms, and paralysis, without wasting, in the legs. But in central tumours the pains in the arms are usually much slighter than in the thickening of the membrane, and there is rarely the early and limited impairment of all forms of sensation which results from the damage to the nerve-roots in the latter disease. This is also true of syringo-myelia, in which tactile sensibility usually persists, although untypical cases may present much difficulty. The diagnosis is considered more fully in the account of that disease.

The affection of the cord itself which most closely simulates the symptoms of tumour is a very chronic transverse myelitis. In such a case as that shown in Fig. 104, p. 338, the diagnosis may be a matter of extreme difficulty. The radiating pains in this case were severe and local, and the limitation of the symptoms, for a long time, to one side, with crossed sensory and motor palsy, presented an almost perfect analogy to the effects of a growth. But it is very rare for severe and acute radiating pains to result from focal myelitis, although a painful sense of constriction is common; moreover, in tumour, one half of the cord seldom suffers severely without the functions of the other half also being very soon impaired in some degree. The limitation of chronic myelitis to a single focus is also very unusual; a considerable vertical extent of the cord is usually involved in varying degree. Hence the practical difficulty in diagnosis is not so great as, at first sight, might appear. Acute or subacute myelitis can only be confounded with a growth in cases in which inflammation results from pressure, and develops with independent energy and rapidity. In such cases the occurrence of myelitis must be recognised, and the diagnostic problem is the detection of the preceding symptoms of the growth that excites the inflammation. This is usually easy; the severe pains, and commonly also the slighter symptoms of paralysis, which existed before the rapid increase in the loss of power, are sufficient proof of a chronic morbid process, to which the inflammation is secondary.

The early pains are often thought to be neuralgic, but their constancy and continuance should suggest organic irritation; their seat, moreover, is rarely that in which neuralgia is common. The pain produced by tumours of the cauda equina is often felt first in the sciatic area, and thought to be sciatica; but they are commonly from the first bilateral, while sciatica is almost invariably one-sided, and bilateral sciatic pain should always suggest irritation where the sensory fibres from both sides are near together, and can be irritated by a single cause, *i. e.* it should suggest disease within the vertebral canal. In all cases, however dubious the early pains may be, other symptoms soon become added, and prove that there is more than a simple neuralgia. But the effect of the organic disease on other parts of the nervous system may evoke any functional derangement to which the patient is predisposed, and it is most important not to permit the diagnosis to be thereby misled, or the gravity of organic symptoms to be under-estimated.*

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* Thus the patient from whom the tumour was removed (see p. 561) had once had

If the existence of a tumour is clear, two questions remain—its seat and nature. The interference with the elements of the cord shows its lateral seat, but this is of small practical importance compared with the evidence of its vertical position, in any case in which an operation is contemplated. The latter is indicated by the level of the transverse symptoms, especially the upper level of the sensory or motor loss, or sensory irritation; but it is of special importance to note that the growth is often above this, and never, or almost never, lower. It may be even a distance of three or four vertebræ above the pair of nerves corresponding to the highest level of the anæsthesia or pain. This discrepancy is especially noticeable when the growth is in the cord; it is least when the tumour is extra-dural; and if this is so seated as to affect a nerve on one side, there is often a strict correspondence between the symptoms and the situation of the lesion. It is explained by the upward course of many posterior root-fibres.

The nature of the tumour is also of great practical importance in many cases. If the patient has had constitutional syphilis, the syphilitic nature of the growth is highly probable, and the probability is further raised in degree if the growth developed rapidly, so that the symptoms attained a considerable intensity in less that three months from their onset. The coexistence of a cerebral lesion also increases the likelihood of the syphilitic nature of the growth. Either of the last two considerations suggests the same conclusion in any case in which syphilis, although not proved, is possible; but it must be remembered that cerebral and spinal tumours, of other than syphilitic nature, may coexist. In the same way, tubercular and scrofulous processes suggest the tubercular nature of a tumour, and this is further supported by early adult age, by rapidity of course, by the speedy extension of the symptoms to the second side, by the slightness of evidence of irritation, and, in some cases, by periods of arrest of the progress of symptoms that were slow in their development. Such arrest indicates an arrest of the growth of the tumour, which we cannot infer from the subsidence of symptoms that developed rapidly, and may have been due to secondary inflammation. When a rapid onset is due to this cause, irritation symptoms are usually pronounced. Tumours elsewhere always constitute strong evidence of the nature of one in the cord, but such an indication is rarely available, since the spinal canal is rarely the seat of secondary growths. Multiple tumours are

some convulsive attacks, and the intense pain due to the growth having caused slight mental symptoms, I was asked to see him to decide the question of the hysterical nature of the affection. The fact is peculiarly instructive from a diagnostic point of view. Not less so is a case of central glioma in a girl (sent to me by Mr. P. B. Mason, of Burton-on-Trent), in which a central growth in the cervical region extended up into the medulla, and caused at last universal palsy. The first symptoms immediately followed severe mental disturbance; to this they were naturally ascribed, and thought to be functional. A similar sequence is met with in cerebral tumours (*q. v.*).

probably either sarcoma or neuroma. In the absence of these indications a meningeal tumour is probably a sarcoma, perhaps fibrous or myxomatous, and one within the cord is probably a glioma, because these are the most frequent growths in those situations. A diagnosis founded on mere frequency of occurrence has necessarily only a low degree of probability, and must be sometimes wrong; but it may be highly probable that the growth is one of two or three kinds, and this may be of considerable practical value. To the surgeon the point is of great importance.

Prognosis.—Unless the tumour is syphilitic, or can be removed, the prognosis is necessarily most grave. Of all syphilitic lesions, growths are those that are most amenable to treatment, and the effects of which most certainly pass away, if they have not reached an extreme degree or lasted too long a time. If, however, the damage has been allowed to remain unchecked for several months, recovery may be imperfect. Tumours of most other kinds steadily increase, and the resulting damage is on the whole steadily progressive, although its course may be varied by stages of rapid (myelitic) increase in the symptoms, followed by stationary periods and even by transient improvement. In the case of growths outside the cord, the prognosis has been rendered much less gloomy by the progress of surgery.

Treatment.—If the growth is syphilitic, appropriate treatment should be energetic and prompt, so as to effect some diminution of the pressure as quickly as possible, since the longer this continues the more considerable are the degenerative changes, and the longer these will endure. Even the influence of a few days before treatment becomes effective may make a difference of weeks in the duration of symptoms, and lessen the degree of ultimate recovery. Whenever the symptoms suggest a tumour, and syphilis is possible, it is most important that suitable treatment should be employed; it can do no harm if the tumour is not syphilitic, and may save the patient's life and strength if it is. Syphilis can only be excluded when there has been no possibility of infection. In many cases of late syphilitic lesions there is no history of secondary syphilis, and in others there is no history of a primary sore. Hence it is *a priori* certain that in some cases of late lesions a history of both primary sore and secondary symptoms will be absent, and, as a matter of fact, such cases are met with not unfrequently. Of course these cases cannot be counted as syphilitic in any scientific investigation into the influence of this disorder, but in treatment we have often to allow weight to considerations which are insufficient to influence scientific investigation, and treatment determined by the mere possibility of syphilis is often justified by its results. Tubercular tumours are also amenable to treatment to some extent, but they are too rare to be of much practical importance.

In most other cases we can do little more than treat symptoms, relieving pain by sedatives, watching the state of the bladder, preventing the occurrence of cystitis, and guarding the patient from bedsores. We must remember that sedatives will probably be needed for a long time, and they must therefore be used as sparingly as possible, lest custom and tolerance deprive them of their power. Cocaine may be used in many cases as an aid in economising the influence of morphia.

But many tumours within the spinal canal may be removed, at any rate if they are outside the substance of the cord. The anticipation that such growths would be found removable was expressed in the first edition of this book (1886), in words that may be quoted because they were soon afterwards realised:—"Modern methods render the opening of the spinal canal far less formidable than it formerly was, and the removal of a tumour from the membranes of the cord would involve less immediate danger of serious consequences than the removal of a tumour from the brain."

On the other hand, growths within the spinal cord are in a very different position in regard to surgical procedure, since the removal of such a tumour could hardly fail to cause a traumatic inflammation that would damage and perhaps destroy all the elements of the cord at the spot, and would probably be permanent in its effects, especially when these structures have already suffered from pressure. At the same time the early removal of a small growth might possibly be followed by the regeneration of conducting fibres that are on the other side of the organ, and the return of their function, lost only through the effects of pressure.

In a man with complete paraplegia, motor and sensory, of slow development, accompanied by attacks of agonising spasm, a diagnosis of tumour led me to advise the removal of the growth. This was done by Mr. Horsley, and the result has been the permanent recovery of the patient. Although the intense spastic paraplegia showed that there must have been complete degeneration of the pyramidal fibres, every indication of this has passed away. The tumour was a myxo-fibroma, about the size of a split almond, which had so compressed the cord in the upper dorsal region as to reduce its thickness to about one half. It was within the dural sheath, and adherent to the cord.* It is certain that many growths might be removed without difficulty, and with a secure result, although only one instance has been since recorded. In this, the tumour was a lymphangioma cavernosum, situated outside the dura mater, over the cauda equina.†

In connection with an operation, a question sometimes arises that is of very great importance, and often of equal difficulty. The symptoms are consistent with the syphilitic nature of the growth; and this cause and nature cannot be excluded, except by the disproof afforded by the inutility of treatment. But it is essential that this disproof should be afforded before an operation is decided on; and the question arises, how long should be allowed for this process of practical exclusion, —how long should treatment be continued without result to justify the last resort? It needs to be longer when the symptoms are of considerable duration than when they are recent, since secondary changes of long duration cannot be quickly removed. Time is needed in every instance to permit a trustworthy conclusion, but a month is probably always enough to enable a result to be reached that will justify either further delay on the one hand, or immediate procedure on the other.

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* The details of the operation are full of instruction in reference to similar procedures, and are given in the account of the case in the 'Med.-Chir. Trans.,' vol. lxxi, 1888. The surgical aspect of the subject is too large to be entered on here.

† By Laquer, of Frankfort-a.-M., 'Neur. Cent.,' 1891, p. 193.

Finally, one injunction given by Mr. Horsley may be emphatically endorsed. If it is clear that the growth is not syphilitic, and that no good can be done by other treatment, delay in an operation can only cause harm—can only result in a less favorable state for the proceeding, less chance of recovery, less degree of recovery, longer and greater suffering, and should on every ground be avoided.

CAVITIES AND FISSURES IN THE SPINAL CORD

(SYRINGO-MYELIA, HYDROMYELIA, HYDRORRHACHIS INTERNA).

Cavities in the spinal cord, distended with liquid, are met with at all ages, and those which present certain features, and often cause certain symptoms, are now known by the above terms. The following figures illustrate the most important facts regarding this morbid state, and its somewhat complex conditions will be best understood by considering the appearances presented in these, and their significance, as described in the explanations.

The term "Syringo-myelia" is applied to all cavities in the cord with well-defined limits; but the majority of these (with the exception of such as exist in tumours of manifestly different nature), whatever their condition or form, or the changes they have undergone, are surrounded by a layer of embryonal neuroglial tissue, and outside this the white substance of the cord is deficient in amount. Wherever met with, or whatever the secondary changes the tissue presents, this fact of environment stamps the cavity or cavities (for there may be two) as congenital, because this abnormal material is due to a persistence of the embryonal tissue from which the cord is developed. The cavities are thus due partly to defective closure of the tube which is first formed (residual portions of which become distended by accumulation of liquid), and partly to the breaking down of this persistent embryonal tissue or to tissue formed from it by a process of growth. Moreover the cord sometimes suffers serious damage from pressure, owing to the distension of the cavity by the liquid which accumulates within it, or by growth of the adjacent tissue. The condition is not rare, but is often overlooked, especially in its slighter forms, and is readily misinterpreted when it is associated with acquired disease—as in the condition illustrated in Fig. 105, p. 339, which is very instructive in this connection. The term "Hydromyelia" has been applied to the simpler forms,* in which the cavity is merely the dilated central canal; but as there is no real difference between this and other varieties, the designation, being unnecessary and even misleading, is falling into disuse.

To understand the origin of these conditions it is necessary to remember the mode of development of the spinal cord. The sides of the primitive furrow of the embryo coalesce, so as to form a canal of relatively large size, the walls of which are thinner in front and behind than at the sides, and consist of elongated cells, some of which are arranged regularly at the inner surface of the cavity, like an epithelium. The anterior wall first becomes thickened, to form the white commissure and the front part of the grey, while an abundant growth of cells occurs at the sides of the tube, and extends forwards (*i. e.* downwards in the position of the embryo), thus forming the lateral and anterior columns; between these two projections is the depression of the anterior fissure. Next occurs a growth backwards of the now thick sides, which forms the posterior parts of the lateral columns and the adjacent part of the posterior columns, and with this the posterior roots are connected; this forms the "root-zone" of the posterior columns. The posterior median columns are formed later. The central canal extends backwards between these two rudimentary postero-external columns, being closed behind only by a thin layer of cells. As the posterior columns increase in thickness the posterior part of the canal becomes narrowed, and its walls unite near its anterior extremity so as to form the posterior grey commissure, and divide the canal into two parts. Of these the anterior becomes the permanent canal, while the posterior is reduced to a narrow fissure between the posterior columns. Ultimately this posterior narrow part becomes closed by a growth of cells which occurs from behind forwards, as the median parts of the posterior columns develop. All these parts consist first of embryonal cells, which afterwards undergo a transformation into the nerve-elements proper, and the order of transformation is the same as that in which the parts were formed, the posterior median columns latest. The cells of the grey matter are formed before the fibres of the white columns. Some of the embryonal elements undergo a different and slighter change. They may almost be said to persist, constituting the neuroglia, and the gelatinous grey substance continuous with it around the cord, and also massed at the posterior root fissure and around the central canal.

The central canal ultimately lies in the anterior part or middle of the grey commissure. It may persist through life as a cavity, circular in section, or as a slit, antero-posterior or transverse, and is lined with epithelium (Fig. 172). Around the canal there are usually many nuclear elements, especially on each side of it, and these frequently fill up the lumen of the canal, so that its position is indicated only by an oval mass of small cellular elements (Fig. 172, D). The obliteration of the canal often occurs quite early in life, and is no evidence of any morbid process. The canal may be found obliterated in one part of the cord, and patent in another (C, D, Fig. 172). The simple closure of the canal by nuclei does not usually cause any distension of the part which is above the closed portion, or, at any rate, not more than a very slight increase in size, so trifling that its significance is open to question.

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* See Leyden, 'Virchow's Archiv,' Bd. lxviii, p. 1.

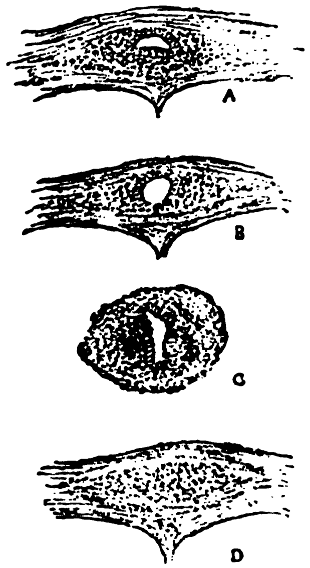

Fig. 172.—Central canal in normal cords. It has the form of a transverse slit in A, a vertical slit in C (from the conus medullaris), while in B it is circular. In each it is lined by columnar epithelium and surrounded by cellular elements, rounded, or angular from compression, mingled with granules. In D, which is from the same spinal cord as C, the position of the canal is occupied by a mass of nuclear tissue.

Syringo-myelia occurs chiefly in the cervical and upper dorsal regions of the cord. The cavity is often closed below by gliomatous tissue, and the excess of this may extend up into the medulla and even pons. Occasionally the cavity exists through the whole length of the cord.

In the simple condition found in young children, or in adults when no secondary changes have occurred, the morbid change presents the two features already mentioned — a quantity of tissue of translucent aspect, which, from its structure, is clearly persistent embryonal tissue in which nerve-elements have not developed, and a cavity due to imperfect contraction of the canal. The change is always confined to the posterior half of the cord, and usually to the posterior columns, which, as we have seen, are formed last. The persistent embryonal tissue may be evidently increased by a process of various degrees of growth, and the cavity enlarged by distension. Further, when the persistent tissue is considerable in quantity it may break down, and thus give rise to a new cavity, or enlarge that which has developed from the primitive canal. Thus the cavity may be distinct from the canal, and in some cases the abnormal tissue around it is very small in amount, and reduced even to a mere wall. It may also be separate, because it is the hinder part of the embryonal canal, cut off by coalescence of the walls.

These conditions are shown very well in the adjoining figures, after

Leyden (Figs. 173, 174). In the first, at A, the cavity presents nearly the shape which it has at one period of development, before the formation of the posterior columns is completed, and without the separation into two parts produced by the formation of the posterior commissure. It is bounded by a narrow layer of embryonal tissue, which widens out behind, having nearly the shape of the posterior median columns, which are, as we have seen, the last to be formed. In B and C, the cavity is much larger, and the posterior columns are smaller, either by an earlier arrest of development or by greater distension, or, most probably, by the influence of both these agencies. The cavity extends up to the posterior surface of the cord, and is closed in there only by a narrow layer of cellular structure.

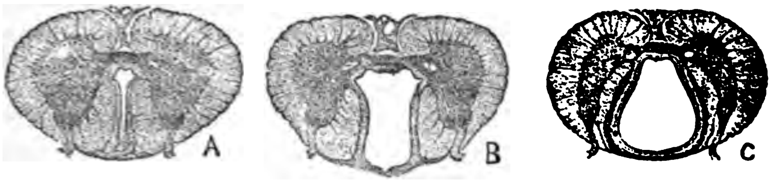

FIG. 173.—Syringo-myelia, from a child two years old, with encephalocele and absence of cerebellum. (After Leyden.) In A, cervical region, the central canal is large, lined with epithelium, and from it a medial fissure extends backwards nearly to the periphery of the cord, limited by a layer of homogeneous tissue, more abundant at the posterior limit. In B, lower down, the fissure is enlarged to a cavity of considerable size, while in C it is still larger, and the posterior columns are reduced to a narrow zone between the layer of tissue which bounds the cavity and the grey substance.

In Fig. 174 we have somewhat more complex conditions. The cavity in the first section closely resembles in appearance that in A of the last figure, but differs in the important fact that the posterior commissure has apparently been formed and the central canal developed in the normal manner, although it has become obliterated by nuclei. This is clear, if the commissure is compared in the several figures. The cavity, therefore, does not, as in Fig. 173, represent the whole of the primitive canal, but only the posterior portion after its division. The adjacent tissue is much more abundant than in the other case. The projection into the posterior extremity of the cavity indicates a process of active growth, which is also shown by the irregularity of the cavity in B, the large amount of tissue, and the wide separation of the posterior horns. In C, from the lower part of the cord, the embryonal tissue occupies two oval areas in the posterior column (by mistake, clear in the figure), while the cavity is closed or nearly so. (Lower down, a cavity existed in the centre of each of these areas, apparently due to breaking down of the tissue.) In D, again, the embryonal tissue occupies only the middle line, as a wedge-shaped area, due to the defective formation of the medial part of the posterior columns. It has been mentioned that the neuroglia may be regarded as a persistent, slightly modified embryonal tissue, and these exuberant masses of tissue bear considerable resemblance in structure to gliomata, so that the condition has been called "gliomatosis." In each of these cases the brain participated in the defect of development; there was internal hydrocephalus, and the cerebellum was absent.

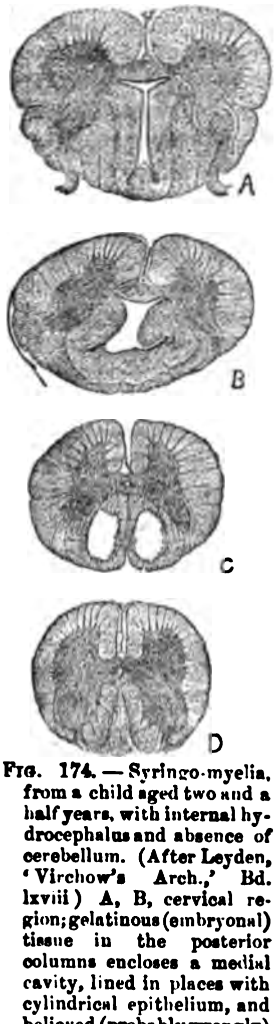

FIG. 174.—Syringo-myelia, from a child aged two and a half years, with internal hydrocephalus and absence of cerebellum. (After Leyden, 'Virchow's Arch.,' Bd. lxviii) A, B, cervical region; gelatinous (embryonal) tissue in the posterior columns encloses a medial cavity, lined in places with cylindrical epithelium, and believed (probably wrongly) to be the central canal. C, lower dorsal; in each posterior column is a mass of similar gelatinous substance (which, by a mistake, is shown in C as an open cavity). In D, lumbar region, this tissue occupies only the position of the posterior median columns.

The cavities in the adult cord present many varieties. The most common form resembles in its essential features the congenital disease which we have just considered. Between the two there are also differences, but the resemblance is sufficiently close, and the origin of the differences is sufficiently clear, to make it practically certain, as already mentioned, that the two forms are really the same, and that the form met with in adults is the congenital condition, persisting until later life. Of the characters which this form has in common with the congenital disease, the most important are the position of the cavities and the presence of tissue in their vicinity of embryonal or gliomatous nature. The differences depend on an apparent increase of this tissue by a process of growth, on the greater damage to the other parts of the cord by the distension of the cavity, and on the extent to which the formation of new cavities or enlargement of old ones takes place by a process of disintegration of the newly formed or persistent tissue. The damage to the cord produced by distension of the cavity involves most the nearer and less resisting parts. The grey substance, therefore, suffers in greatest degree; the white columns to an extent that depends in part on the precise position of the cavities, as presently to be described. The latter also present, in many cases, the complicating changes of secondary degeneration, ascending or descending, as shown in Fig. 175, due to the compression of certain tracts at some specially affected spot.

As the simplest condition, we may have a dilatation of the central canal surrounded by gliomatous tissue disposed as in congenital cases. This tissue is often more abundant, and forms a more distinct mass, lower down the cord, below the cavity. A good example of this condition in slight degree is presented by the cord, the seat of chronic myelitis, represented at Fig. 105, p. 339. In the cervical region (A) there is a large central canal, bounded by a thick layer of gliomatous tissue. In the inner part of this tissue, limiting the cavity, is a sinuous membrane, fibrous in structure. The origin of this membrane is not easy to explain, but it is often found in cavities of this character, and is evidence of the similar nature of those that differ much in other respects. In the lumbar region (B) the tissue, which bounds the cavity in the other section, forms a large round mass in the position of the canal, obliterating it.

When a central cavity appears not to be the canal itself, but to be situated behind, it is then apparently often due to a persistence of the fore-part of the posterior portion of the canal, after the posterior com-

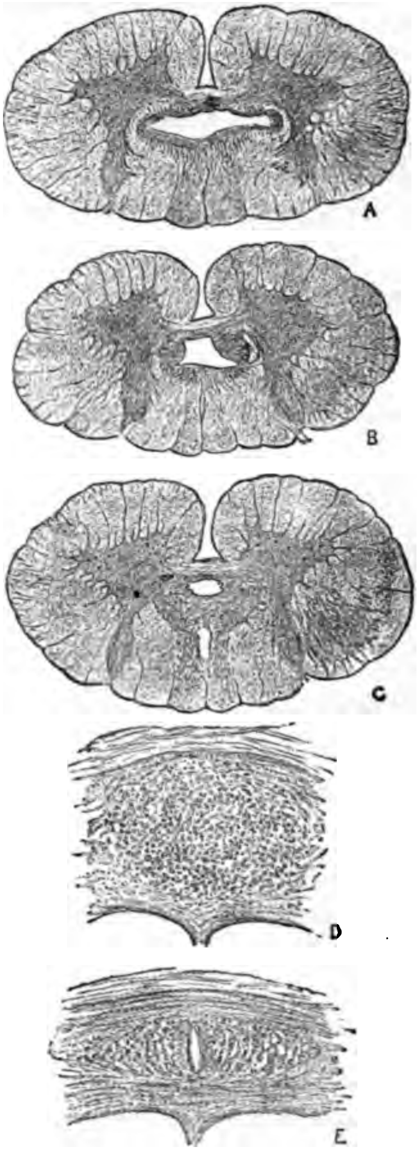

FIG. 175.— Syringo-myelia, from a case of tumours of the pons and cauda equina, with some diffuse sclerosis in the dorsal region of the cord. A, B, and C are sections of the cervical enlargement. The large cavity in A is perhaps the dilated canal, since it is lined in front with epithelium, but in front of it, in the posterior commissure, is a group of nuclei like an obliterated canal. The zone of tissue around the cyst is composed of interlacing fibres and nuclei, and processes extend from it into the posterior columns, both in front and behind. In B, the cavity is smaller and the wall thicker, while in C, the tissue about it forms a considerable mass, and a second small cavity has formed in the middle line, apparently by the breaking down of the tissue. In the dorsal region (D) there was merely an unusually large accumulation of nuclei in the position of the canal, which, in the lumbar region, had the normal aspect (E). The cord presents degeneration of the right pyramidal tract (descending from the tumour in the pons), and of the post. med. cols. and ant. lat. ascending tract, probably ascending from the sclerosis.

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missure has been formed. In Fig. 175, for instance, we have a central cavity, but in the commissure in front of this is a mass of nuclei having the usual aspect of an obliterated canal.[*] The cavity is surrounded by a zone of tissue, which in A and B sends off a fringe of processes, especially backwards. This tissue is increased in the lower part of the cervical region (C). In B, a small cavity is formed between the tissue and the grey matter by breaking down, and in C another exists in the middle line behind the growth, probably due to deficient closure of the original fissure, and increased by a destructive process. Still lower, the commissure resumes its normal appearance, but the collection of nuclei in the position of the canal is unusually large (D). In the lumbar region, the canal is patent (E). The degeneration of the posterior median column and of the lateral columns is secondary.

We have seen that in the congenital cases we must recognise a process of growth of the persistent embryonal tissue, to account for some of the conditions met with, and a like process is suggested by the central mass in Fig. 105, B. Hence it is not surprising that in many cases the growth should attain the dimensions, and assume the characters, of a positive tumour. In most cases the tumour has been central in position, and has had the structure of a glioma. It has occupied a large part of the area of the cord at a certain part.[†] In the chapter on the tumours of the cord it has been pointed out that sarcomata also may grow from the tissue around the central canal, as in the case shown in Fig. 170. Hence it is not surprising that the condition of syringo-myelia, even if this is congenital in origin, should be frequently associated with definite tumours. The growths have often been multiple, apparently the result of a wide-spread tendency, and perhaps connected with a wide-spread persistence of embryonal tissue, of which remarkable instances are sometimes met with. In the case shown in Fig. 175 there was a tumour of the pons and also one of the cauda equina. The nature of the growths is uncertain, but they were probably either sarcoma or glioma. The same tendency to morbid growths is illustrated also by Fig. 176, which is similar in many respects to that just considered. In this case also there was a tumour of the pons and one of the cauda equina, and there was also a central growth in the dorsal region, occupying the greater part of the area of the cord, to which the ascending degenerations were secondary. In A there is a large central cavity which is probably not the central canal, since, as in the last figure, the position of this is marked by the oval group of nuclei in the anterior part of the grey commissure. The zone of tissue which bounds it is narrow, but a few processes are given off behind. In B, first dorsal, this tissue forms a compact mass, and the processes are numerous and very similar to those in the last figure. In C, the commissure is reduced almost to normal conditions. In this cord we have also an instance of the fact that cavities may occur in other parts, very similar in their general characters to those that occur in the central region. In B, a large cavity occupies the intermediate grey matter and posterior cornu on the right side, with some morbid tissue bounding it on the medial side. In C, the cavity is smaller

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[*] This is the probable interpretation. At the same time it is possible that the nuclei have not this significance, and that it is really the central canal. It is often more difficult than might be imagined to say whether a cavity does or does not represent the central canal. The presence or absence of an epithelial lining has generally been taken as a criterion, but it is doubtful whether this has any significance. Around the whole of the original cavity the inner layer of cells is arranged as an epithelium, and if any part of it persists it is probable that epithelium will persist also. Moreover the epithelium often disappears from the wall of the dilated central canal itself; always where it is enlarged by breaking down of tissue.

[†] As in an interesting case described and figured by Riesinger, 'Virchow's Archiv,' Bd. xcviii.

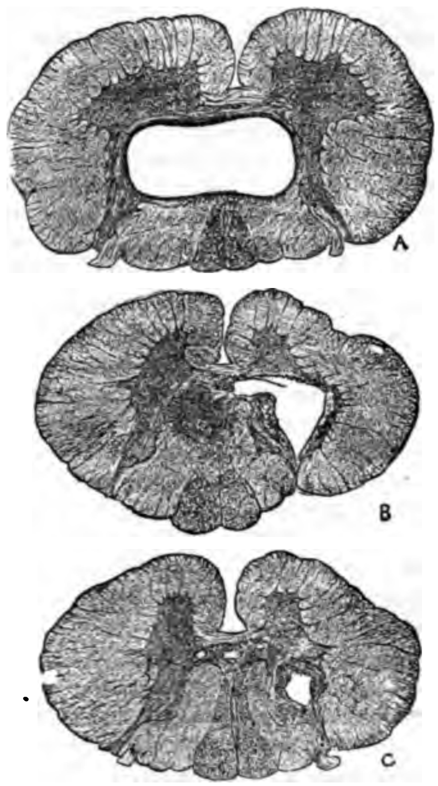

FIG. 176.—Syringo-myelia, from a case in which there were tumours in the dorsa region of the cord, the cauda equina, and the pons Varolii. A, mid-cervical region. A large oval cavity lies behind the grey commissure, surrounded by a narrow wall, chiefly composed of fine fibres, but with a wavy membrane on the inner surface. In front of the posterior commissure, an oval group of nuclei has the aspect of an obliterated central canal. A few short processes extend from the wall of the cavity into the posterior columns. There is secondary ascending degeneration of the post. med. cols., right direct pyramidal tract, and ascending antero-lateral tract in all the sections. B, first dorsal. Behind the posterior commissure is a growth consisting of small cells, round and fusiform, the latter with round nuclei. It contains many vessels with thickened walls. A fringe of fine processes extends from it into the posterior columns. An irregular cavity occupies, on the right side, the posterior half of the grey matter, which is reduced to a narrow layer around the cyst. This is lined by a delicate nucleated membrane, outside which is a thicker membrane, lying in folds, the section of which is thus sinuous. Outside this again is a small-celled growth similar to that behind the commissure. In C, a little lower, this cavity is smaller, the sinuosities of the wall greater, and the growth outside it more abundant. (Still lower the area was entirely occupied by the growth.) In the posterior commissure there is only a quantity of loose nucleated tissue in the situation of the growth.*

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* For the opportunity of drawing these sections I am indebted to Dr. Dreschfeld. A full account of the case has been published by Dr. Harris in 'Brain,' Jan., 1886. The large growths were sarcomata.

and the tissue around it is more abundant, but easily distinguishable from the grey substance, while a little lower down the cavity disappeared and the tissue formed a compact rounded mass. The cavity is lined by a delicate layer of cellular membrane: outside this is a fibrous membrane, lying in folds which appear as sinuosities in the section. The existence of this membrane shows that the cavity is not simply formed by the breaking down of the tissue. The tissue is quite similar in structure to that which is behind the commissure in B.*

The posterior cornu is not an uncommon seat of cavities, which may extend through the whole length of the horn. An example of this is shown in Fig. 177. Hæmorrhage had occurred into the cervical region of the cord (A), which was enlarged and distended with blood, so that the precise character of the cavity in this region was indistinguishable; the blood also filled the cavity through the

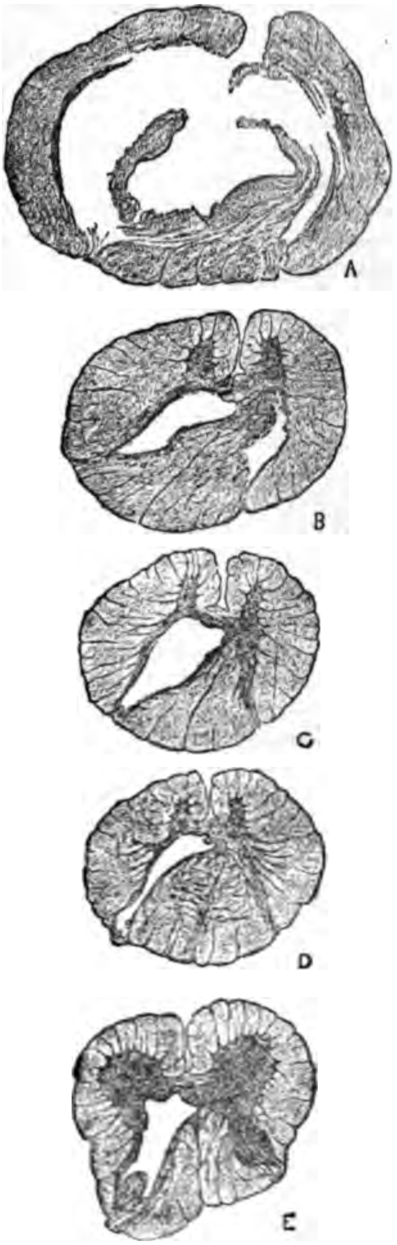

FIG. 177.—Sections of a spinal cord in which a cavity existed throughout its length. Hæmorrhage in the cervical region was the immediate cause of death. The extravasation had there distended the cord (A), and had burst into the cavity, which was filled with blood. A smaller cavity existed in the right cornu, in the upper dorsal region (B). In most parts the inner surface was slightly irregular, as if produced by breaking down of the tissue, but near the position of the central canal a sinuous membrane existed in places, without, however, any epithelial covering.

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* In connection with the tumour of the cauda equina in these two cases, it may be mentioned that the same coincidence has been observed in other instances. There was a tumour in that situation, for instance, in the case described by Riesinger, who, rightly regarding the condition as congenital in origin, suggests that the tumour may be connected with the fact that the embryonal cord occupied the entire length of the canal.

entire length of the cord. In the upper dorsal region (B) there are two cavities, one in each posterior cornu, but that on the right ceased a little lower down, while the other extended to the lumbar region, as shown in the figure, reaching almost to the surface of the cord. The upper and inner extremity of this cavity is in the position of the central canal, no other trace of which exists. There is some abnormal tissue in the neighbourhood of the commissure, and also in C, where irregular tracts pass transversely from the median septum into the fore-part of the posterior columns. The presence of this tissue, and the relation to the central canal, suggest a congenital origin. During the reduction in size of the canal, in the development of the cord, the cavity presents at one period short lateral processes (compare Fig. 179, A), and we can therefore understand how a still earlier arrest of development may lead to the persistence of a lateral process on one side or both, surrounded by residual embryonal tissue, and that the cavity should afterwards extend in this tissue or in the grey matter by a process of growth and disintegration, such as probably had occurred in Fig. 177, D. A comparison of D and E suggests that the two cavities were united in the middle line in the upper part of the cord, where the hæmorrhage took place.

In the conditions we have hitherto considered, definite limited regions of the cord have been involved through a considerable vertical extent. The changes are sometimes, however, much more diffuse in their distribution, as is shown by the remarkable lesions represented in Fig. 178. In this cord, which is that of an adult, we have a combination of the same two conditions of abnormal tissue and cavities. Scattered through the nerve-substance are tracts of a peculiar tissue, represented by the darker shading of the figure, and in many regions thus affected there are cavities, for the most part fissure-like, as if the cord had been split here and there. At first sight the appearance suggests an active and recent morbid process; nevertheless I think that the congenital origin of the condition is beyond doubt. It will be observed that the morbid changes, as in the other forms of syringo-myelia, are chiefly in the posterior half of the cord; small areas of disease in the anterior columns, in the sections E and F, are the only exceptions. The minute structure of the abnormal tissue was exactly like that of the normal gelatinous layer beneath the pia mater. Besides the conspicuous areas figured, it could be traced here and there, in small tracts, through the whole of the posterior columns and the hinder parts of the lateral columns, and also in the posterior horns, in the grey matter of which it could be readily distinguished. The relation of the cavities to this substance is distinct; where they extend beyond it, this is apparently the result of the distension of the cavities and an actual fissuring of the cord, along the lines of connective tissue, by the pressure of the contained liquid. The medial cavity in C is doubtless due to defective closure of the original fissure between the posterior columns. In the lumbar region the nerve-fibres are absent from the front of the posterior columns, where a coarse network of this residual tissue encloses empty spaces.

The damage to the grey matter may entail alterations in the related nerves, and even, in cases of long duration, in those connected with the posterior roots. In the peripheral nerve-fibres of the skin and muscles, changes of the ordinary chronic degenerative character were found by Déjérine, in cases in which sensation was altered or the muscles wasted.

Thus these various forms of syringo-myelia, in spite of their variations in seat and form and general aspect, when closely studied, seem all to be connected with gliomatous tissue; and that the morbid state takes its origin in abnormal conditions during the development of the cord is practically certain from their seat and features. This lesson is clearly taught by the facts shown by the cases here illustrated. It is probable, therefore, that this conclusion is also true of cavities of similar character and position in which no gliomatous tissue can be recognised; and most of the hypotheses regarding the origin of such cavities from processes of simple myelitis and the like have no sufficient foundation. But we are still ignorant of the mechanism by which the defect in the process of development is brought about. It has been extensively ascribed to an inflammation of the embryonal neuroglia at an early stage of development, but of this there is no real evidence. It is, moreover, no explanation to assume a primary intra-uterine hyperplasia of the embryonal tissue. It is, indeed, possible that more than one morbid process during the early stage of development may cause the condition, and that closure of the canal below may have consequences during development which it has not at a later period. But it is important to recognise the fact that there is a gradation between the chief varieties, and that some are associated with developmental defects in the brain, to which they can only be related as the common expression of a defect in the developmental tendency of the germinal tissue; and what must be true of some forms is probably true of most. This may seem only to push the difficulty into the region of the inexplicable, but it is only placing the origin of the malady where we are compelled to refer the causation of so many congenital diseases, which, after all, bear but a small proportion to the physiological characteristics that spring from the same source.

The symptoms of the disease have followed some illness, mental shock or anxiety, and traumatic influences, but it is probable that in no case have these done more than excite the manifestations of pre-existing disease, or induce local changes in the morbid state. The possible influence of injury has certainly been over-estimated, since in some of the cases in which it seemed effective the malady was unquestionably congenital, and the traumatic influence only terminated its latent stage.*

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* Among the chief papers in which the subject will be found discussed are those of

Symptoms.—The manifestations of syringo-myelia depend on the secondary processes of distension and growth, and the damage thus produced to the functional elements of the cord.[*] Hence the state is sometimes discovered after death when it has given rise to no symptoms, and its existence has not been suspected during life. In young children, indeed, it appears never to cause any definite symptoms, while the disturbance of function is variable in occurrence and degree—much more so than in character. The central position of the disease entails considerable uniformity in the distribution of its local consequences,

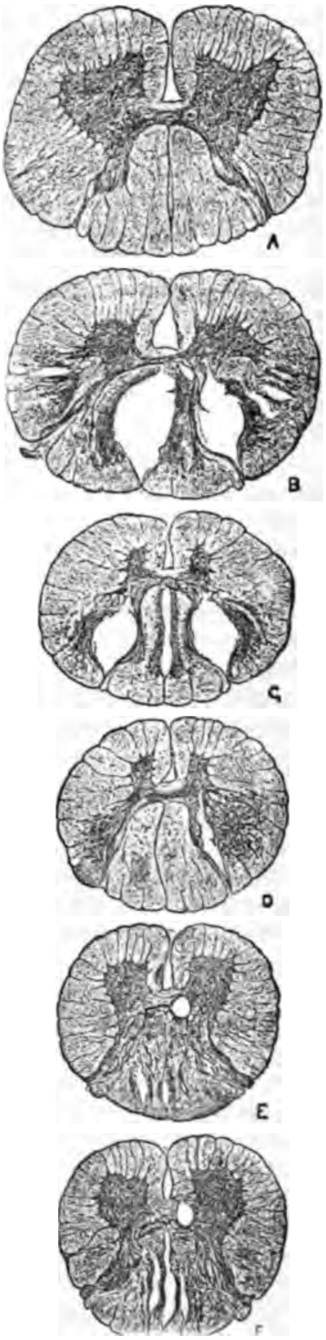

Fig. 178.—Cavities in the spinal cord. A, mid-cervical, presenting merely some increase of connective tissue in the left lateral column. In B there are several cavities having the aspect of distended fissures, the largest in the middle line of the posterior columns and the right posterior cornu; smaller ones in the lateral columns. Adjacent to each is a quantity of dense tissue, which had a peculiar and uniform structure. It consisted of a very fine network of fibres with minute nuclear bodies in the interstices. No larger cells were observed. Throughout the posterior and lateral columns this tissue could be seen, here and there, thickening the neuroglia, where no conspicuous masses were formed. It could also be traced in the posterior cornu, and was readily distinguished from the spongy substance by its slighter staining. The distribution of this tissue is shown in the other sections by the darker shading. In C, the fissure in the middle line is narrow. In E and F, the anterior part of the posterior columns contained a coarse network of this tissue, and no nerve-fibres. The central canal was obliterated, but the group of nuclei that marked its position could be traced throughout the cord.

Schultze (see below); Hellich, 'Arch. Bohême d. Med.,' 1890, iii, No. 4; Kronthal, 'Neur. Centralbl.,' 1888, p. 333; Oppenheim, 'Charité Annalen,' 1886, p. 409.

[*] They have been fully described by Schultze, 'Zeitschrift f. klin. Med.,' 1888, p. 525; and 'Verh. Congr. Inn. Med.,' 1886; and by Allen Starr, 'Am. Journ. Med. Science,' 1888, vol. xcv.

and hence also in the derangement of function that results from simple increase in the lesion either by distension or growth. Hence the symptoms are often such that they have considerable diagnostic importance, although in a minority of cases they are less uniform in character, probably because the disease is unsymmetrical, or irregular in position or extension, whether the symptoms are due to the distension of a cavity, or to this combined with adjacent growth of tissue. It then often causes symptoms indistinguishable from those of a tumour of neoplastic nature. Moreover, the precise level at which the chief morbid process takes place, leads to a corresponding variation in the position, and therefore in the general aspect, of the symptoms it produces. The fact that the lesion is almost always greater at the cervical region causes the arms to be the most common seat of its manifestations, while the occasional invasion of the fourth ventricle by the associated growth, and the occurrence of coincident secondary changes in this part, explain the involvement of some of the cranial nerves that has been met with in a few instances.

The symptoms are usually of slow development, increasing gradually in the course of years. Their main features are twofold, of which one is clearly and certainly intelligible, while the explanation of the other is a matter of hypothesis, although also not difficult to understand, at least in part, if the opinions expressed in the chapter on the functions of the cord are correct. The two leading features are loss of sensibility, chiefly to pain and temperature, and muscular atrophy, resembling in characters that met with in the "progressive" spinal form. Of the two, the sensory loss is the earlier and more constant. The muscular atrophy is a necessary consequence of the damage to the anterior cornua from the compression they endure, when and where the central cavity or adjacent growth attains such a size as to damage them seriously, and they may be reduced to a mere zone between the cavity and the outer ring of white substance. The destruction of the motor nerve-cells involves the degeneration of the nerves and the wasting of the muscles. The loss of sensation is intelligible if the path of painful impressions is by the posterior commissure to the antero-lateral ascending tract. Although the fibres of this tract that lie in the periphery, and have crossed far below, may be expected to escape (and the sensory loss is not usually met with in the legs or lower part of the trunk), the enlargement of the central canal and disease in its vicinity can hardly fail to damage early and much the fibres that cross at the level of the chief lesion, and constitute the path from posterior roots not very far below. The course of thermal sensations is still quite unknown; the only fact we have—the frequency with which such sensibility and that to pain are involved together—points to contiguity of path, and prevents surprise at the special affection of this form of sensibility in syringo-myelia. Tactile sensations are lost only in rare cases; our ignorance of their path prevents us from explaining the fact, but we know, from other facts of disease, that it is distinct from that for pain. Moreover, the occasional localisation of the morbid process in the posterior columns, and even cornua, may explain the involvement of all forms of sensibility, and also its occasional restriction in area, since it may be due to direct interference with the posterior root-fibres after they have entered the cord.

In many cases the affection of sensation of pain and temperature is equal; in others the latter is lost to a greater degree and extent than the former. Its loss is often the earliest symptom, as the scars of burns may show. The loss to heat and cold may not correspond. Thermal or painful hyperæsthesia has preceded the loss, as with other forms of sensation when destroyed by processes that slowly damage the fibres, and irritate before they interrupt. Perversion of sensation has been noted, heat being felt as cold, and *vice versâ*. Spontaneous sensations are common, doubtless from the irritation of the fibres in the early stage, or of their upper portions when they are interrupted—feelings of heat or cold, and in some cases pains, various in character, sometimes paroxysmal like neuralgia, or sharp and brief like those of tabes, and occasionally referred to the joints, or felt chiefly in some part adjacent to the seat of the other symptoms. Pain in the spine may be complained of, and is, perhaps, a direct effect of the disease. Often the malady causes no suffering. The loss of sensation is usually sharply limited, and the arms or upper half of the trunk is its common seat. It is usually found in the same part as the muscular wasting, but is more extensive, especially below the seat of atrophy. Thus, when the muscles of the shoulders and upper arms only are wasted, the sensory loss may be in the forearms and hands. This we should expect from the fact that the crossing of the sensory path is some distance above its entrance into the cord, while the motor roots arise from nerve-cells mostly near their level of exit. Exceptions may be due to the affection of the root-fibres, as explained above. Further, the fact that the distension and damage always extend through a considerable vertical extent of the cord makes the relation of the two sets of symptoms more extensive and less regular than if the disease were limited to a small region.

The "muscular sense" is said to have been generally normal when tested. Occasionally there has been unsteadiness on standing with the eyes closed.

The motor symptoms usually come on after the sensory loss, long after in slight cases, and consist in muscular weakness and wasting in the upper limbs; while, if the legs suffer, it is generally from simple spastic paralysis, such as would result from, and is no doubt due to, compression of the pyramidal tracts by the disease in the upper part of the cord. It is rarely complete. The unsteadiness of movement, above mentioned, we may connect with the tendency of the lesion to involve the posterior columns, and so probably to interfere with the path from the muscles to the cerebellum. Rarely there is atrophy of the legs similar to that of the arms, and in such cases the disease extends into the lumbar enlargement. Reflex action in the legs varies according to their state; it may be normal or increased; the knee-jerks are often augmented, with rectus- and foot-clonus, or, in the cases last mentioned, myotatic irritability may be lost—very rarely on one side only. A curious tremor of the limbs has been noted in some cases.

The muscular atrophy in the arms begins in, and affects most, certain parts, according to the position of the chief damage to the grey matter of the cord. It may be in the hand and region of the ulnar nerve, or in the muscles supplied by the musculo-spiral and radial nerves, or in the shoulder and upper arm muscles.[*] It is a slow wasting, with gradual lowering of the electrical irritability, as in progressive muscular atrophy, although, as in that disease, some groups of muscles may present indications of the reaction of degeneration in the early stage of their affection. Fibrillation is common. As the wasting and weakness increase in the part first affected, they spread to others, to the rest of the limb, and to the adjacent parts of the trunk. Weakness of the trunk muscles often leads to lateral curvature of the spine; and the convexity, owing to posture, is usually to the left. The occurrence of similar symptoms in the legs is extremely rare. Both sides usually suffer together; a unilateral affection has indeed been observed, but is altogether exceptional. The sphincters may escape or be involved.

Trophic disturbances in the extremities affected with sensory loss are not uncommon. The skin may become thin and glossy, or thick and horny. Acute changes may occur, eczema, herpes, or bullæ; whitlows; deep, obstinate ulceration, and even gangrene. More frequent, however, is simple vaso-motor disturbance, coldness and lividity of the extremities, or redness with swelling and heat, and sometimes a tough œdema, which may be local like a tumour, and afterwards pass away. The nails share the trophic changes in the hands, becoming grooved, fissured, and cracked; they may even drop off. The secretion of sweat is lessened in the part, as was well marked in one unilateral case.[†] The action of pilocarpine has been found to be delayed, although ultimately increased (Grasset). The bones may become thickened or brittle, and joint changes, like those of tabes, sometimes occur.

It is rare for the symptoms to extend into the region of the cranial nerves, although in occasional cases various disturbances of function have occurred, presumably due to the extension of the changes into the medulla and pons, and chiefly met with when there is upward extension of the associated growth, already mentioned. Thus there have been paralysis of one vocal cord, the tongue, and of the face, difficulty in swallowing, disorder of respiration and of the heart's action. The organs of the special senses usually escape, with the exception of the eyes, in which certain disorders may occur, probably due to disease of the path of the sympathetic in the cord, or to that of the fourth ventricle. Thus the pupils are often unequal, the smaller being on the side of the more severe cord symptoms, on which the fibres for the radiating muscle of the iris are likely to be damaged. Narrowing of the palpebral fissure and slight ptosis have been also noted. Nystagmus is not rare.*

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[*] Blocq thinks that spastic paraplegia is chiefly met with in the cases in which the atrophy begins in the region of the ulnar nerve, and tabetic symptoms in the legs when it begins in the radial area; but the data for the generalisation need to be more extensive.

[†] Allen Starr, 'Am. Journ. Med. Sc.,' May, 1888; Rumpf, 'Neur. Cent.,' 1889, p. 257.

The course of syringo-myelia is, as a rule, slow, and measured by years, although an apparent exception is presented by the cases in which it is associated with spreading overgrowth of the adjacent gliomatous tissue, or acute destructive processes occur in this and spread to its vicinity. In such cases a considerable development of the disease may be attained before symptoms are produced, and these may rapidly reach a considerable degree, and cause death in a few months or even less. In most cases death is the result of exhaustion, of the impairment of the functions of the medulla, or, more commonly, of some complication—bedsores, cystitis, and the like.

Diagnosis.—The resemblance of the disease to more common maladies led to its confusion with them during life, until the careful observations of the last few years have established features by which its existence can often be at least surmised. The most important characteristic is the combination of muscular atrophy with still earlier sensory loss, involving temperature and pain far more than tactile sensibility. But the occasional implication of all forms of sensation must be kept in mind, and also the fact that in slight and slow or anomalous cases, the sensory symptoms may long predominate, and for a time exist alone. The position of these symptoms (in the arms), and their frequent combination with spastic palsy in the legs, is a further characteristic—not, however, unequivocal.

Cervical pachymeningitis causes symptoms which, in general character and in distribution, alike in the arms and the legs, very closely resemble those of syringo-myelia; but the affection runs a more rapid course, the anæsthesia involves all forms of sensibility, and has not a greater but a less extent than the muscular wasting; the two correspond more closely in their seat; pain is a far more prominent symptom, and the reaction of degeneration is common in the wasting muscles. Tumours of the spinal cord only cause symptoms of like character, and especially of similar symmetry, when they are central in situation, and the diagnosis may then be very difficult and even impossible. But the resemblance is often more than superficial, for such growths generally arise from residual gliomatous tissue, and are such as are frequently associated with syringo-myelia. It is chiefly from the more rapid development of symptoms, and their more speedy extension, that such a growth can be suspected; and only the presence of slighter chronic preceding symptoms of the character above described would justify the expectation that a cavity is associated with the growth.

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* Contraction of the fields of vision, especially for colours, has been observed, but is not easy to explain (Déjérine and Turland).

Progressive muscular atrophy is sufficiently distinguished by the absence of impairment of sensation, and in peripheral neuritis the pains, mode of onset, and degenerative reaction in the muscles should preclude any danger of mistake. In "Morvan's disease," to be presently described, the symptoms may bear a close resemblance to those of syringo-myelia, and it is possible that they are due in part to a similar condition of the spinal cord. As a rule, however, the early loss of tactile sensibility, and the greater prominence of trophic disturbance in the tissues, especially the painless whitlows, enable the diagnosis to be made. There is only one acute affection of the cord that enters into the diagnostic problem, and that is spinal hæmorrhage. It has been mentioned in the account of this condition that the hæmorrhage is sometimes into a pre-existing cavity—as in the case shown in Fig. 177. The presence of a cavity into which the blood has been effused may be suspected if the hæmorrhage has been preceded by any symptoms suggestive of syringo-myelia, inquiry for which should always be made; or if a rapid extension occurs through a considerable vertical extent of the cord, distinctly abolishing its central functions.

The malady, however, varies much in seat and characters, as is evident from even a cursory glance at the illustrations given above; the symptoms, of necessity, vary with the lesion. The diagnosis may be difficult and even impossible in such cases, especially in the early stages and slighter forms, when irritation and pain predominate, and even neuralgia may be simulated. It may be necessary to wait, for time to show the course of the symptoms. Their progressive increase, in spite of variations, should always be taken into account; it frequently is the first feature that suggests the nature of such a disease, contrasting, as it does, with the tendency of inflammatory affections to arrest and improvement. A peculiar difficulty is presented by the cases, not few, in which the first symptoms follow some influence—an illness, a blow, or even mental disturbance—that seems to be their cause, and suggests disease of very different nature. Often, then also, it is only by waiting and watching that a right opinion can be formed. When bulbar symptoms come on rapidly, the nature of the case can only be inferred from previous spinal symptoms.

The PROGNOSIS and TREATMENT of syringo-myelia are subjects on which, unfortunately, its nature permits little to be said. If its existence can be recognised, the malady is generally advanced, and its observed rate of progress is the only ground on which a forecast can be based. When the central functions of the cord are considerably impaired, and the symptoms are steadily increasing, it is rare for life to be prolonged for more than one or two years, and the subacute onset of considerable paralysis may herald a course that leads to death in a few months. On the other hand, a stationary condition may last for many years. Treatment can do nothing for the morbid state, nor is it likely that the progress of spinal surgery can afford the means of relief. The severity of some of the individual symptoms may, however, be lessened by appropriate measures; especially trophic changes may be diminished by care and early treatment, bedsores may be prevented, cystitis guarded against, and if any acute paralysis occurs, the possibility of some recovery may suggest the maintenance of the irritability of the muscles by electricity, which cannot be expected to influence the slower atrophy. Pain may need the customary sedatives; but early pain, especially with the hyperæsthesia that depends on the intensification of the nerve-impulses at the irritated part, may be much lessened by the diminution of these by cocaine, used as in tabes. Sudden pain in the spine may indicate commencing hæmorrhage, and should lead to the adoption of the posture and other measures appropriate to that grave affection.

Analgic Panaritium; Morvan's Disease.

By the term Morvan's Disease is generally known an affection that was called Panaritium Analgicum, or "Painless Whitlows," by the physician of Brittany, Morvan, who, in 1883, first described it.* Observations on it are still few,† but, judging from two fatal cases,‡ it appears to consist in a combination of a condition of syringo-myelia, or of the associated gliomatosis, with a peripheral neuritis in the extremities. The symptoms make the constancy of the neuritis probable; we must wait to learn how far the morbid state of the cord is constant, and whether the changes in the nerves take origin in developmental conditions. It is quite possible that they may share the condition of arrested development, entailing a liability to later morbid changes, especially in view of the analogous conditions observed in the case mentioned on p. 469.§ At the same time, it is possible that the symptoms of this affection may be due to local neuritis of various nature, and we must be cautious in inferring from them that the pathological state is the same in origin in all cases. In some instances the malady has been apparently set up by an injury. The affection has been met with in both sexes, but more frequently in males, and has generally commenced during the first half of adult life, between twenty and forty. When local injury has preceded the symptoms it has sometimes been at a considerable interval (Häckel), and it has been unilateral when the latter have been bilateral, so that its relation to them is uncertain.

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* 'Gaz. Hebd.,' 1883, Nos. 35—44.

† The chief writings are those of Louazel, 'La Maladie de Morvan,' Paris, 1890; Joffroy and Achard, 'Arch. de Med. Exp.,' 1890; Charcot, 'Prog. Méd.,' 1890, Nos. 11 and 12; Häckel, 'Münch. med. Wochenschr.,' 1889.

‡ Gombault, quoted by Charcot; Joffroy and Achard, 'Arch. de Méd. exp.,' 1891.

§ In a case recorded by Jolly ('Charité Ann.,' xvi, 1891) there was also a congenital anomaly of the fingers, a web of skin between the third and fourth fingers of each hand.

The characteristic symptoms are in the upper extremities; only in rare cases do the feet suffer at a late period. Neuralgic pains may occur first, but the definite symptoms consist of weakness and muscular wasting in the hands and forearms, loss of all forms of sensation, and especially a peculiar trophic change—whitlows on the fingers, with recurring deep ulcerations in various parts of the digits; those near the extremity of the fingers may only heal with the loss of some of the terminal phalanges. The loss of sensibility involves all forms, and usually precedes the occurrence of the whitlows, so that these are painless—a peculiarity which constitutes the most salient feature of the disease, and suggested, in the first instance, its special character. Occasionally the trophic disturbance precedes the anæsthesia, and the whitlows and ulcers are then painful. The latter are deep, and often resemble the perforating ulcers of tabes; cracks in the skin accompany them, and the nails shrivel and split. Vaso-motor derangements, lividity and pallor, often precede and accompany the disturbance of nutrition. The progress of the affection is very slow, and extends over many years; one hand is usually affected some time before the other. The electrical irritability of the nerves of the part has been found normal in the early stage, and slowly vanishing as the malady progressed. Although the trophic changes are limited, as a rule, to the hands, and the muscular wasting does not extend above the forearms, the loss of sensation is occasionally more extensive, involving the whole arms, parts of the trunk, and even the face. An affection of the shoulder-joint has been observed.* The feet are occasionally, though rarely, the seat of anæsthetic ulceration; the legs have also been found weak, with excessive knee-jerk and foot-clonus (Häckel).

In the autopsy that was made by Gombault neuritis was found in both arms, and also a diffuse overgrowth of connective tissue in the posterior part of the grey matter of the cord and the posterior columns. This growth involved also the coats of the vessels, which were in places so thickened as to obliterate their cavity. Neuritis has also been invariably found in the extremities of the fingers when these have dropped off.

The features of the malady are so peculiar that, if they are known, the nature of a case cannot well be mistaken. Anæsthetic leprosy presents the closest resemblance, but in this the peculiar ulcerations are absent, and pigmentary alterations occur in the skin; while in Raynaud's disease there is not the peculiar loss of sensibility, nor an equal tendency to whitlows, and vaso-motor disturbance is a more conspicuous feature. In the peculiar malady known as "sclerodactyla" (a form of scleroderma that affects the fingers and face), there is no loss of sensibility or tendency to destruction of the finger ends. Simple syringo-myelia is distinguished by the common absence of the whitlows, by the subordination, in time and degree, of the trophic changes to the other symptoms, and by the common preservation of tactile sensibility. But the distinction is probably not an absolute one. In Morvan's disease there is peripheral neuritis as well as syringo-myelia, and to this is due its special features, and especially the early and intense trophic disturbance.*

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* Morvan, 'Gaz. Heb.,' 1887, No. 34.

SPINA BIFIDA.

Spina Bifida, or split spine, depends on a defect in the closure of the vertebral arches, which usually leads to the protrusion of the membranes as a sac, forming an external tumour, into which the lower part of the spinal cord often extends, normal or variously altered in conformation, sometimes even reduced to a neural lining of part of the wall. Rarely, there is no external prominence—a form known as "spina bifida occulta."† The condition is most common in the lumbar region of the spine; sometimes is present at more than one place, very rarely in the whole length of the vertebral column. It is met with in about one child out of every thousand born (Chaussier). The disease is one of chiefly surgical interest, and therefore only an outline of the more important facts is here given. The spinal cord, whether normal or not, generally reaches lower than usual in the vertebral canal, as it does at the early period of development, when the morbid state is produced,—becoming adherent at its lower extremity and remaining so. In many cases the central canal of the cord is enlarged above, and the lower part of the cord may also be similarly distended.

The precise condition presents many variations, which can be, in part at least, understood by what is known of the process of development, a subject that has been very ably discussed by Bland Sutton. The essential element in spina bifida, the defect in the vertebral arches, depends on a deficiency in that ingrowth of the mesoblast from each side which should enclose the embryonal spinal cord, and form bone, after the superficial epiblastic layers have united to form the epidermis. A similar ingrowth of mesoblast forms the corium, muscle, &c. This apparently takes place in excess in spina bifida occulta, and leads to a curious condition, usually present—an excessive growth of hair over and about the affected part, which is generally the lumbo-sacral region. On the other hand, in many cases in which there is a tumour, this superficial mesoblastic ingrowth seems to be deficient, as well as that for the vertebral arches, so that, at the upper part of the tumour, there is an area in which there is no proper skin, but a peculiar shiny membrane destitute of hair. It is probably this superficial mesoblastic deficiency that permits the protrusion, and formation of an external tumour.

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* The absence of any fundamental difference between this malady and syringomyelia has been pointed out by Bernhardt ('Verh. Vereins inn. Med.,' Berlin, January 19th, 1891) and Jolly (loc. cit.). But the additional element of neuritis must not be ignored.

† The best accounts of spina bifida are in the "Report of the Committee of the Clinical Society" ('Transactions,' 1885), and Bland Sutton's "Lectures on Evolution in Pathology" ('Lancet,' February 25th, 1888). Spina bifida occulta is described by the same writer in the 'Lancet,' February 25th, 1887.

In spina bifida occulta the defect in the arches can be felt on palpation, either in the lumbar or sacral region, and the spinal cord extends lower than normal, as in the ordinary form. The latter, in which there is an external tumour, presents three chief varieties, according as the sac contains only the spinal membranes (meningocele), the spinal cord as well as the membranes (meningo-myelocele), or the latter distended by enlargement of the central cavity (syringo-myelocele). These are the chief classes; they do not, indeed, exhaust the rarer forms, but to take full cognizance of these would entail a very complex list of varieties.*

Of the several forms, that without an external tumour has been least frequently met with, although it is probable that a knowledge of the significance of the growth of hair in the lumbar region would lead to the detection of this state in many cases in which it is now undiscovered. Putting this form aside, simple meningocele and

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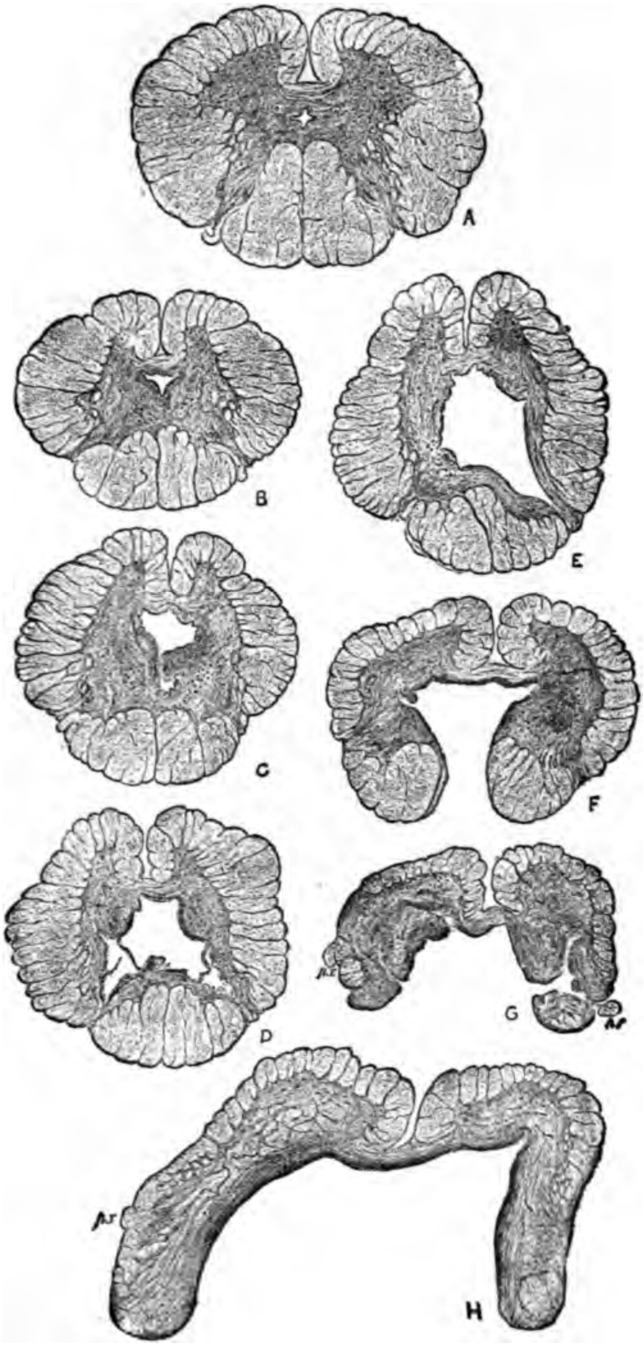
* Several other varieties are enumerated by Bland Sutton (loc. cit.).

FIG. 179.—Spina bifida: sections of a spinal cord, from a case the symptoms and history of which I was unable to ascertain. In A, cervical region, the only abnormality is a large cruciform central canal and an unusually thick grey commissure. This continues through the dorsal region, at the lowest part of which there is a great change. In B, the canal is larger, and the necks of the posterior horns (and posterior vesicular columns) are united by much commissural tissue; in this, many vertical fibres could be seen, and others running from before backwards in the middle line. In C, an extension of the canal backwards has taken place, and it is bounded by a sinuous membrane: the outer part of the grey substance is atrophied. In D, the cavity has extended into each posterior horn almost up to the surface of the cord, in part by a breaking down of tissue, because the membrane limiting the enlarged canal remains undestroyed. In E, still at the junction of the dorsal and lumbar regions, the cavity has receded from the left horn. In F, the division of the cord has taken place, not in the direction of the cavity in the horn (which is filled up), but in the middle line, at or close to the median septum. In G, a wider separation of the posterior columns has taken place, and a cavity has formed on the right side, which extends into the horn, and almost cuts off the posterior column. In H, the cord is spread out and formed part of the wall of the sac. The posterior nerve-roots (*p r*) mark the position at which the posterior horn comes to the surface. The grey matter is in many parts atrophied and translucent, and, in H, contains many large vessels. The septal lobulation on the surface of the cord is greater than normal.

Fig. 179.



syringo-myelocele are both rare; the common form is meningo-myelocele, in which the cord, altered or intact, extends within the sac. The Clinical Society's Committee found that this was the condition in 62 per cent. of the cases in which there was an external tumour. The lower part of the spinal cord is generally adherent to the posterior wall of the sac, where its traction often causes a depression on the surface, always at the membranous area, which, as already mentioned, is generally to be observed in the upper portion. The cord may again become free, and extend downwards in the cavity, or it may be flattened, expanded, and lost in the wall, of which its tissue really forms an inner layer. In this layer there is no distinction of grey and white substance. In either case, the lower nerves arise in the wall, and pass, first in this, and then forwards, across the cavity, to their foramina of exit. The arachnoid always extends into the sac, and the fluid is that contained within the subarachnoid space; sometimes there is an external opening from which the fluid flows.

As a rule, the central canal of the cord is not continuous with the sac; and it may be closed, even in the condition of syringo-myelocele, in which the canal is dilated. In other cases the canal opens into the cavity, sometimes by only a small aperture, even when the lower part of the cord expands into the wall, and the nerves course along the wall in a layer continuous with the arachnoid and superficial to the membrane that represents the cord-tissue from which they arise. As stated, ordinary syringo-myelia may exist in the upper part of the cord. The extension of the cord in the wall of the sac probably indicates a developmental defect similar to that of the bony canal; in the lumbar region the primitive canal has failed to close, so that the cord is open posteriorly, and the two posterior columns may even be far apart, or the cord may even be applied, in the form of a thick or thin lamina, to the wall of the sac.

An instructive although rare example of the involvement of the cord in the developmental effect is shown in Fig. 179, which illustrates also the manner in which the latter may involve in some degree the whole cord. In the cervical region the grey commissure is unusually large, and the canal is cruciform, a shape which it presents at one period of development, and which is here persistent. This condition obtains throughout the dorsal region until, in the lower part, the tissue behind the commissure is so abundant as to unite the necks of the posterior horns. Below this, the canal enlarges into a condition of syringo-myelia, surrounded by the abnormal tissue which, as we have seen, is commonly associated with such enlargement; the canal is limited, moreover, by the sinuous membrane that is so often met with (see p. 567). The cavity extends also into the posterior horns, clearly by a process of disintegration, since in D the membrane lining the enlarged canal persists, and marks the limit of the canal and the extent of the simple disintegration. The progressive separation of the posterior columns is shown in F, G, and H. In the lowest part of the cord, the development of the posterior columns has been apparently hindered, as will be observed if G be compared with F. In the former there are also some fissures in the grey substance, formed apparently by disintegration. The grey substance of the cord is conspicuously wasted in the lumbar region, thin and translucent.

In rare cases there has been no proper development of the cord. It was represented by a mere nerve-like string in a child, who also had hydrocephalus, but lived five weeks. Another child of the same parents had a similar condition.*

Symptoms.—If the cord is intact, symptoms may be absent. In the cases in which there is an external opening, and a constant escape of cerebro-spinal fluid, the child usually dies in a few weeks. In other cases the defective development of the lower part of the cord, or its distension in syringo-myelocele, is manifested by an absence of function, amounting to complete paraplegia in severe cases, which also seldom live. Some cases, however, survive, with paralysis and wasting of the legs, and often absence of power over the sphincters. In slighter forms the paralysis and wasting have been partial, either existing from the first, or supervening at some later period, probably from the occurrence of damage to the nerve-roots or cord-tissue by increased tension, or by inflammation; of the latter abundant traces may be found after death, and various external influences may readily set it up. Some children, whose legs have appeared natural, have never been able to stand; possibly the defect in the posterior columns has given rise to a sort of congenital tabes. Even in the slighter forms, including spina bifida occulta, paralysis and atrophy have come on after some years, and even after adult life has been reached. Indeed, in the unsuspected forms without a tumour, there may be special danger of injury to the unprotected structures, and damage to the nerve-roots. The muscular paralysis and atrophy have been usually below the knees, and the muscles least prone to suffer are the tibiales antici. Hence talipes varus tends to occur. These muscles may alone persist, probably from the higher position of origin of their nerves (Remak). Another peculiarity of these cases is the tendency to perforating ulcer in the feet, which has several times necessitated amputation. Chronic changes in the tarsal and metatarsal bones have also been met with, somewhat like those of tabetic arthropathy.† The ulcers suggest neuritis, which was found in one case; and, in this and others, the arteries have presented thickening of the muscular coat, even leading to their closure. These symptoms may be greater on, or confined to, one side, and on this the knee-jerk has been lessened.‡ These points of resemblance to tabes may also be

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* S. Wolfe, 'Phil. Rep.,' June 2nd, 1883.

† In one case Sutton found the compact tissue very scanty, and the bones filled with semi-fluid fat; a deep ulcer reached a carious spot.

‡ Details of cases by Fischer, v. Recklinghausen, Brunna, and others, are quoted by Bland Sutton (loc. cit.). See also Delafosse, 'Thèse de Paris,' 1874; Remak, 'Berlin. kl. Wochenschrift,' 1885.

the spine, are caused by a sudden contraction of the spinal muscles in some violent effort, a "rick of the back," as it is popularly termed. It is possible that, in such cases, the primary damage is sometimes to the vertebral ligaments and articulations, and that the cord suffers secondarily. The symptoms may only come on a few hours after the injury in such cases, as if effused blood compressed the cord, or the tension upon it and slight damage set up a graver inflammation. Thus a soldier, in a drunken quarrel, was extended by four comrades, two pulling on his feet and two on his arms, while a fifth sat on his back. No immediate effect followed, but the next morning there was complete paralysis of both arms and legs, which very slowly passed away.

PATHOLOGY.—The anatomical lesions in cases in which the vertebral column is not injured vary much in different cases. Hæmorrhage is often found, sometimes outside the dura mater, sometimes on the inner surface of the membrane, in the pia mater, in the substance of the cord itself, very rarely into the central canal.* Occasionally the substance of the cord has been found lacerated when the vertebral column has not been injured. In many cases there is local softening, commonly yellow in tint, sometimes mingled with red, often involving the whole thickness of the cord, and occasionally extending, as central softening, through a considerable vertical extent. Under the microscope, the usual products of inflammation are seen, sometimes with hæmatoidin crystals. Such softening may occur rapidly in severe local concussion, and be found complete a few weeks, or even a few days, after the injury.† On the other hand, in some cases of complete paralysis, no lesion of the cord has been found, either with the naked eye or the microscope, a few days after the injury.‡ In other instances, in which the cord is examined some weeks or months after the accident, the signs of chronic myelitis are found, in scattered foci or more diffuse tracts, in the white columns or grey substance. The nerve-fibres are wasted, and the connective-tissue elements are increased in quantity, and, in the early stages, there may be a leucocytal infiltration about the vessels, dilatation of the capillaries, and minute extravasations, although none may have been visible to the naked eye. In the anterior cornua the motor nerve-cells may be damaged, sometimes swollen and vacuolated, or shrunken, and the anterior root-fibres may be degenerated. The grey matter is especially apt to suffer when the enlargements are injured; in the dorsal region the change may be confined to the white columns. Ill-defined cavities may exist in old cases, where the nerve-elements have perished. The usual ascending and descending degenerations may be found above and below the most damaged parts. Occasionally there are indications of meningitis, diffuse or disseminated, and sometimes confined to the dura mater.

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* As in a case reported by Chucau and Wickham, 'Prog. Méd.,' 1887. Probably it is only when the canal is previously dilated that this occurs, as it may, without serious injury to the nerve-tissue.

† Edmunds, 'Brain,' vol. vii, p. 103; Obersteiner, 'Wien. med. Jahrb.,' Bd. iii, 1879; Lochner, 'Bayer. Aerztlich. Int.-bl.,' 1857, No. 42; Fromüller, 'Memorabilien,' 1870, No. 12. In the last, described further on, softening was found thirty-two hours after the injury.

‡ Fischer, 'Deut. Zeitschr. f. Chirurg.,' 1883, Bd. xix.

Symptoms.—The effects, immediate and remote, of injuries of the spinal cord extend over almost the whole range of symptoms of cord-disease, and their variations in character and course are almost infinite. It is therefore neither practicable nor necessary to do more, in this place, than to describe their general characters. According to the difference in course, we may divide them into three classes. (1) Those in which the injury causes immediate and severe paralysis, due to instant damage to the spinal cord, the consequence of its laceration, compression, or concussion. (2) Those in which there are at first either no symptoms or only trifling disturbance of function, but in which grave symptoms come on a few days or weeks after the injury. (3) Those in which there are no early symptoms, or only slight and transient disturbance, but at the end of one or several months, symptoms gradually come on, often such as indicate disease of some definite system of structure of the cord, degenerative in nature.

(1) The first class, in which the injury causes instant and considerable disturbance of function, includes the cases in which the cord is directly injured, and also some in which there is no visible sign of damage if the patient dies within a few days. In the former class there is often a manifest lesion of the spine, and the cases are, at least at first, purely surgical in their practical relations. In the latter, the mechanical influence has apparently abolished the function of the nerve-elements. If such patients live longer, either quick recovery ensues, or local softening from disintegration of the fibres that are most damaged. It is an interesting fact that concussion may thus derange function. The absolute integrity of structure on early microscopical examination shows that the result is not due to any minute vascular lesion. The effect has been compared by Reynolds, not inaptly, to the demagnetisation of iron by a blow. Doubtless the influence is exerted on the molecular nutrition of the nerve-elements, and the possibility of recovery, or the subsequent structural disintegration, depends upon the degree of nutritional damage.*

The symptoms, in these cases of severe and immediate effect, are generally those of complete impairment of function. When the injury is direct and partial, such as a hemisection by an incised wound, or a partial bruise of the cord by a spiculum of bone being driven against it (as in the case mentioned at p. 197), the effect may be a partial (*e.g.* one-sided) derangement of function; but in most other cases there is abolition of all the conducting functions at the level of the injury. There is complete paraplegia, motor and sensory, with loss of power over the sphincters. The symptoms are thus those of a total transverse lesion at the affected level (see p. 226). When the cervical region is injured, the arms are necessarily affected according to its seat. Such cases often illustrate very clearly the relation of arm-function to the cord.* The fibres for the sympathetic are often implicated, especially those for the iris, causing inequality of the pupils. Reflex action below, abolished at first by shock, usually soon returns and becomes excessive, but remains absent if the lumbar enlargement is damaged, or is invaded by hæmorrhage; it may be again lost by the downward extension of secondary inflammation, which is equally effective when partial (in the posterior columns or grey matter) as when total, and is more difficult to detect in the former case.† Loss of consciousness is occasionally produced by an injury which does not directly involve the head, as in the case mentioned on p. 590; vomiting is very common at the outset. The cases of direct injury often run a severe and rapid course; and when the secondary inflammation spreads downwards, or the lower part of the cord is injured (as is frequently the case), the tendency to trophic changes in the skin, and to the occurrence of cystitis and pyelo-nephritis, is very great. Œdema of vaso-motor origin, and effusion into joints, may be present in the early stage. If the cord has been directly injured in its whole thickness, survival beyond a fortnight is rare. When it is damaged indirectly, with secondary softening—in what may be termed concussion myelitis—death occurs less rapidly, but many patients die at the end of four or six weeks, while in those who survive the first two months slow recovery often occurs. Even in such cases, death sometimes occurs very quickly, but it is possible that there is then a laceration of the cord. A man, whose case has been recorded by Fromüller, was struck on the back, at the level of the third dorsal, by a heavy beam, and had loss of motion and sensation up to the level of the nipples. The palsy of the muscles of respiration increased, and he died from asphyxia at the end of thirty-two hours. At the spot struck the cord was reduced to a pulp for 3½ cm., without any hæmorrhage.

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* These conclusions have been recently formulated more definitely, but without essential extension, by Schmaus, partly from experiments on rabbits ('Münch. med. Wochensch.,' 1890).

The following case illustrates the occasional loss of consciousness, when there is no evidence of any cerebral lesion. A heavy weight fell upon a man's back; he was unconscious for two days, and at the end of that time the legs were completely paralysed, and continued so for three months, with retention of urine. Then, improvement commenced, in the left leg before the right, and in the latter some spasm developed. At the end of six months, he could walk across the room on crutches. Improvement continued, but at the end of two years his condition became stationary, and when I saw him, six years after the accident, he could only walk half a mile; the right leg was still weak in all parts, and a foot-clonus was present in each leg, greater in the right than in the left.

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* See especially Thorburn, 'Brain,' January, 1887.

† This is the explanation of most cases of injury above the lumbar enlargement, with permanent loss of myotatic irritability, or of all reflex action, as in those described by Bowlby ('Lancet,' 1890, i, 1071). Such descending inflammation must not be confused with the descending degeneration in the pyramidal tracts which entails myotatic excess. The very rare exceptions mentioned on p. 229, in which such loss exists with no lesion to explain it, may be associated with the fact that myotatic irritability is sometimes lost in cases of cerebellar tumour. But the fact that superficial reflex action in the legs is never lost unless the lower part of the cord is diseased, is one of the most certain in pathology. Proof of such descending inflammation is given in the chapter on tumours (p. 552, note).

In cases in which the lowest part of the back receives the force of the blow, as when a severe fall ends in the sitting posture, and the sacral region is struck, the nerves of the cauda equina seem often to be specially damaged, and the posterior roots in greater degree than the anterior. The effect is to cause loss of sensibility in the legs, varying in extent and seat according to the part injured, in some instances specially great about the anus, in others extending over almost the whole of the legs, in others again it is chiefly in the sciatic area: all forms of sensation are usually involved. It is generally accompanied by some muscular paralysis and rapid atrophy, with the reaction of degeneration, especially in the muscles below the knees. Rarely no muscular paralysis exists; but there is loss of power over the sphincters in most cases. Reflex action is lost, and trophic changes are readily produced, both on the feet and over the sacrum. The sensory loss is often permanent; the injury to the nerve-roots being apparently too severe to permit recovery. As the case just mentioned shows, when the earlier effects of the injury are survived and improvement sets in, it is remarkable how long this may continue, and how considerable a degree of recovery may be ultimately attained, it may be in the course of three or four years.

(2) In cases of the second class initial symptoms are absent or slight. After the accident the sufferer may not imagine himself injured, and may be able to walk some distance without inconvenience. In other instances there is tingling in the legs, or in all the limbs, immediately after the concussion, sometimes with some weakness of the limbs, more often with a feeling of stiffness. In the course of a few days graver symptoms come on, usually attended with spinal pain and tenderness, sometimes with some stiffness of the back, and often with pyrexia. Tingling in the limbs increases or develops, and is accompanied by weakness, which often goes on to complete paralysis in the course of one to four weeks. The symptoms vary in their character and distribution, according to the position of the morbid process and its extent. In some cases, in which the dorsal region is most affected, the symptoms are those of simple paraplegia, usually with spasm, and sometimes with early contraction of the muscles. In other cases, in which the grey matter of the enlargements suffers, there is scattered

muscular atrophy in the limbs, often with indications of the degenerative reaction. Tremor in the limbs is conspicuous in some instances. A girdle-pain in the trunk, or sense of constriction in the limbs, is very common. The sphincters generally suffer, and the tendency to trophic changes is usually strong. The character and course of the symptoms is that of a subacute myelitis, and the meagre facts of morbid anatomy leave little doubt that this is the common lesion. Thus a lady was severely shaken in a railway collision. She seemed, immediately after the accident, to have suffered no injury, but in a few days paraplegia developed, and from its consequences she died six weeks after the accident. Throughout the dorsal region of the cord I found indications of subacute myelitis, chiefly in the white columns, varying in its extent in different regions, but in most parts considerable in the pyramidal tracts.* Complete motor and sensory palsy, up to the level of the umbilicus, developed in the course of a week, after a fall downstairs in which the back struck successive steps, although the patient was able to walk immediately afterwards. The symptoms in these cases are sometimes unilateral. A man, driving under a low archway, leaned back to save his head, and his spine was pressed suddenly against the sharp edge of the seat-back. He felt but little immediate effect, but in the course of two or three days, complete motor palsy of the left leg came on, accompanied by hyperæsthesia, but without any loss of sensation either in that leg or in the other. Power slowly returned at the end of three months, and was in time perfect. In some cases of this kind the symptoms are due not to direct damage to the cord, but to its compression by inflammatory products outside it, the result of injury to the vertebral column. A clergyman was thrown from his horse, and there was immediately sufficient weakness of the legs to prevent him from walking; this subsequently increased, so that at the end of a fortnight the right leg was completely paralysed, while the left retained considerable power. There was a girdle-pain at the level of the umbilicus, and a bedsore formed, but he slowly improved, and regained the power of standing at the end of eight months. Such symptoms may come on weeks after the concussion, especially when the effects of this are so slight as to be disregarded, and the patient leads his usual life.

When the damage involves the grey matter, the extent of the muscular wasting varies greatly. It seldom affects both arms and legs, but it is usually irregular in distribution, sometimes wide-spread, sometimes limited. Thus a young man fell from a horse and pitched on the head. He was stunned, and on recovering consciousness about two hours later, felt "pins and needles" in both hands and pain in the back, followed by swelling of the neck and difficulty in moving it.

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* In the face of such a case as this, it is superfluous to discuss the question raised by some writers whether or not the cord can suffer concussion. Moreover, it is certain that such an effect as was extreme in this case, may and must often occur in slighter degree.

The tingling ceased, but was followed by a sense of oppression about the shoulders, and persistent pain in the cervical region of the spine. When I saw him, two months later, there was some weakness of the right arm and wasting of the two outer interossei, and of that part of the long extensor which acts on the two outer fingers, with loss of faradic and preservation of voltaic irritability.

Sensory symptoms are prominent in some cases of this class. Besides the pain in the spine, to be presently mentioned, pains are often felt in the legs, various in character; still more frequent are spontaneous sensations of tingling, "pins and needles," and the feelings of defective sensibility described as "numbness." Various forms of hyperæsthesia are also common, with or without such subjective sensations. This may exist in any part, in the soles, feet, thighs, or not infrequently about the genitals. In the latter region, and about the anus, a sense of coldness is often complained of. In these regions there may be a distinct defect of sensibility, but this is slighter and less frequent than in the cases of the first class. Some degree of anæsthesia to touch may be associated with increased sensitiveness to stronger impressions. These sensory disorders may exist alone or be accompanied by motor weakness. Slight unsteadiness or definite inco-ordination is present in some cases, usually resembling that met with in ataxic paraplegia.

In cases which survive the acute stage there is usually slow improvement, which is often ultimately very great. In many cases recovery is incomplete, but improvement goes on for years, as it does in the cases of considerable damage to the cord already mentioned; the ultimate degree attained is commonly much greater than in cases of corresponding character and severity due to other causes than injury. Some permanent symptoms are left, chiefly when the initial derangement of function has been severe, and has lasted for some time.

(3) In the cases of the third class, chronic symptoms slowly follow an injury at an interval usually of some months, and the cases have the aspect less of a traumatic lesion of the cord than of a primary cord disease, the relation of which to the injury is rather an inference from the sequence, coupled with the absence of other causes, than an obtrusive fact, as in the cases of the first and second classes. The symptoms are usually those of a definite system disease of the cord, less frequently those of a disseminated chronic myelitis of irregular distribution, still more rarely those of chronic focal myelitis. Primary spastic paraplegia, ataxic paraplegia, locomotor ataxy, and progressive muscular atrophy are the diseases which most frequently thus result. The fact that an injury is occasionally the cause of these maladies has been already mentioned in the account of their etiology, and the symptoms of such cases so far resemble those that are due to other causes that it is not necessary here to add anything to their history as already given.* We must assume that the shock to the

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* A case may, however, be mentioned in which typical tabes followed slowly a

nerve-elements causes a slow perversion of nutrition, which is only manifested by disturbance of function when it has gradually attained a certain degree. Occasionally, other causes co-operate, capable of producing degeneration. Previous syphilis, or neurotic inheritance, can sometimes be traced, and it is a reasonable assumption that the predisposition thus arising may assist in rendering the traumatic influence efficient, or in augmenting its effects. Intermediate cases connect this class with the last, cases in which we must assume, from indications of chronic inflammation, that definite injury was produced at one or more spots. An instance of this, and also of focal myelitis, was presented by a man with permanent spastic paraplegia and an intense girdle-pain at the level of the umbilicus. These symptoms commenced three months after a jump from a table, and slight unsteadiness, with pains in the legs, connected the jump and the paralysis.

The several consequences of injury, which we have considered as occurring in separate form, are not only connected by intermediate cases, but are also sometimes distinctly combined. Thus an actual and immediate lesion of the cord may be associated with an early and severe increase in the symptoms from the development of subacute myelitis in other parts. The traumatic inflammation, at the spot damaged, often attains an intense degree, and runs a severe course, in consequence of the influence which may give rise to such inflammation apart from a positive lesion; and, in both cases, the damage to the cord may be perpetuated by a degenerative tendency. It is very common, for instance, for an injury to the cord, in those who have had syphilis, to cause acute symptoms which subside, but not perfectly; and the residual disturbance of function may persist, and even increase in the course of years, in consequence of the influence of the previous syphilis in rendering the nerve-elements prone to degenerate. Again, it has been pointed out that the gouty diathesis is an unquestionable cause of myelitis, and often of the grave relapsing and spreading disseminated variety. A concussion of the cord, which causes only slight symptoms, may excite the occurrence of this form of myelitis in persons thus predisposed; the symptoms of the later inflammation may come on months after the injury (the effects of which have generally not quite passed away), and are sometimes excited by some over-exertion, or slight fresh concussion, which would have had no effect on a healthy individual. I have known the symptoms of insular sclerosis, cerebral and spinal, to quickly follow traumatic paraplegia.

One other symptom remains to be considered, which is common to all forms of traumatic lesion of the cord, and is often severe and persistent when other symptoms are slight,—spinal pain. It may continue for years after other symptoms have ceased, and probably depends on a neuralgic state of the nerves of the membranes, or of

fall on the back, recorded by Barbour ('Journ. of Nervous and Mental Diseases,' March, 1891, Case IV).

those of the vertebral column; often, probably, the pain depends on the nerves of the joints and ligaments of the spine. It is essentially a traumatic spinal neuralgia. The pain may be felt at one or more spots; when severe, it often extends through a considerable length of the spine, and sometimes passes up to the occiput. It is occasionally referred to the sacrum, and may there have the character of a sense of weight or more vague discomfort. The pain is associated with tenderness of the spine, usually deep-seated, chiefly developed at the injured part, but sometimes present also at other spots. It may gradually assume the features of a true neuralgia, may occur in paroxysms, and be induced by mental and other influences which do not act directly on the spine, as well as by exertion, posture, and other agencies that may immediately influence the affected structures. This condition is often called "spinal irritation."

The cause of traumatic lesions of the cord often acts also on the brain. A cerebral lesion may occur from the violence which affects the cord; the cerebral symptoms then coexist with those of the spinal lesion, and may mask the latter during the early stage. More common, however, is functional disturbance of the brain, the result partly, perhaps, of the physical concussion, but chiefly of the mental shock which a serious accident necessarily causes. The resulting condition is favorable to the development and persistence of subjective sensory symptoms. Attention, maintained by concern, has a powerful intensifying influence on all forms of nerve-pain, and certainly aids in keeping up the pain in the back, and even the tenderness which follows injuries to the spine. So marked is the influence of "nervousness" on the subjective symptoms, that it has been even maintained that in a large number of cases of concussion of the spine the symptoms are of hysterical origin.* Well-marked symptoms of hysteria are sometimes manifested by these patients. But, on the other hand, it is necessary to avoid the danger of over-estimating the effect of mental influence, and of regarding, as entirely due to this, symptoms which are real, and are merely intensified by attention. The danger is especially great in cases of railway injuries, concerning which an unbiassed judgment is not easy to secure, and in which, when objective symptoms are absent, it is easy to minimise suffering, and attribute too much to the mental condition. The sinister influence of litigation on the intellect may be traced very widely.† I believe that it is rare for symptoms to be purely mental. It is often asserted by those employed for railway companies that subjective symptoms quickly subside when the sufferer's "claims" are settled, but it should be remembered that mental anxiety is a potent cause of diseases of the nervous system, and must be strongly opposed to recovery from genuine disorders. The occurrence of improvement when suspense is at an end, is thus no proof in itself of the nature of the case, and its significance has been unquestionably over-estimated; moreover, in a great many individuals whom I have had an opportunity of observing long after they had received their "damages" (as the expression curiously runs) this subsidence had not occurred, and even the "sovereign balm" of substantial compensation has appeared to do very little for the relief of the sufferer.

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* J. J. Putnam, 'Boston Med. and Surgical Journal,' 1883, Sept. 6th.

† Not many years ago it was customary for the "experts" who gave evidence on behalf of railway companies to deny that the spinal cord could be injured if the legs were unwasted. Although the opinion dare not now be expressed, its significance is not without analogies at the present day.

These opinions have been formed from a study of cases other than those that involve litigation, in which no elements existed to bias the judgment, and from a comparison of these with many "railway" cases observed apart from forensic proceedings. Those who desire to learn what can be said on the subject of "railway spines," as they have come to be termed, when viewed from the opposite sides, will find abundant material for consideration in the writings of Erichsen ('On Concussion of the Spine,' London, 1875) and Clevenger ('Spinal Concussion, or Erichsen's Disease,' Philadelphia, 1889) on the one side, and of Page ('Injuries of the Spine and Spinal Cord,' London, 2nd ed., 1885) on the other. Scattered papers by J. J. Putnam, Walton, Spitzka, Buzzard, and others, more or less instructive, will be found epitomised in Clevenger's work, where indeed is collected the pith of almost all that has been written on the subject.

Diagnosis.—The chief points in the diagnosis of traumatic lesions to the cord have been already incidentally considered. Immediate symptoms may be due to laceration, hæmorrhage, or to simple concussion, and the diagnosis between these is not always possible at first. If there are immediate symptoms of a partial lesion, these indicate direct injury, while the rapid subsidence of the disturbance of function renders simple concussion probable, and excludes any considerable direct injury. The later development of paralysis indicates myelitis, unless there is evidence of considerable irritation of the nerve-roots at a certain level, which suggests inflammation outside the cord, and perhaps even outside the dura mater. The greatest diagnostic difficulty is presented by the cases just mentioned, in which the symptoms are subjective, and anxious attention has been long given to the local discomfort. The chief elements in the diagnosis of these cases have been, however, already indicated. It is important to search for, and to give due weight to, any symptoms beyond the simple spinal pain. Slight "tingling" or "creeping" sensations may be of cerebral and "functional" origin, but a persistent sensation of "pins and needles" rarely is of that nature. A definite sense of constriction is also strongly suggestive of organic disease, and so is a well-marked difference in the power of the muscles on the two sides. The latter is of least significance if the excess is slight, general, and on the right side—of much greater significance if the diminution is partial, and affects only certain groups of muscles, such as the flexors of the hip and knee, or the peronei. Any impairment of power over the bladder or rectum is of great diagnostic importance; loss of sexual power, on the other hand, is of little value, since this function is readily depressed by mental anxiety and preoccupation. A slight change in reflex action is most significant when it is partial. A foot-clonus, or rectus-clonus, is strong presumptive evidence of organic mischief. A slight excess of the knee-jerk is of little value; although it probably always indicates some changes in the nutrition of the spinal cord, it does not indicate structural disease. In all cases it should be remembered that the absence of any common symptom is of far slighter significance, as evidence of integrity of the cord, than is the presence of that symptom as evidence of disease. It may seem superfluous to insist on a consideration so elementary, but it is still possible, as experience proves, for a medical witness to assert in a court of law that a claimant's spinal cord cannot have been injured because some symptom is absent, the presence of which would be important.

Prognosis.—Immediately after an accident a cautious prognosis should be given, even if the symptoms are slight, on account of the possibility that grave disturbance may develop in the course of a few days. In developed cases, the prognosis must in general be guided by the same considerations as those which determine our estimation of the probable course of symptoms of similar character and severity due to spontaneous myelitis. To this there are, however, two general exceptions. First, the danger of death, if any exists, is greater in traumatic cases than in others, as long as the symptoms are increasing. Secondly, if there is no danger to life, or such danger has passed, the prospect of improvement is distinctly greater than in a case of similar features but of non-traumatic origin. If the symptoms are slight or moderate in degree, approximate recovery may be anticipated, although slight symptoms often endure for a very long time. Indeed, in many cases, recovery, although approximate, is not perfect. The patient is never quite as strong, never becomes quite as capable of exertion, as before the injury. A cautious prognosis should also be given whenever there is the late and gradual onset or increase of symptoms that suggests a degenerative process. Such degeneration presents far less tendency to arrest or subsidence than do the earlier lesions. As a rule, the sooner symptoms occur, the better is the prospect of ultimate improvement or recovery, provided they are not so severe as to be incompatible with life.

Treatment.—The early treatment of these cases, and much of the later treatment of those in which the spinal column is injured, is purely surgical. The points of medical character alone need special mention, and many of these have been anticipated in the preceding pages. In all cases in which spinal symptoms are present immediately after an injury, however slight those symptoms may be, absolute rest should be insisted on for some days or weeks, according to the severity of the early symptoms. This is necessary on account of the secondary inflammation, which, as we have seen, so often occurs. The treatment of developed symptoms must be conducted on the same general principles as in cases of myelitis; the details need not be here repeated. If there is muscular wasting, it is important that the nutrition of the muscles should be maintained by electrical stimulation, since a very considerable amount of ultimate recovery may be anticipated, and it is important to keep the muscular tissue as far as possible in a condition to respond to the nerve-power when this returns. If there is reason to believe that there is inflammation of the membranes, or inflammatory effusion outside the cord, compressing it, mercury may be given, but this condition is probably much more rare than might be anticipated. The influence of mercury on inflammation of the substance of the cord is doubtful. The chief element in treatment is patiently to permit time to do its work, and the tissues to slowly regain such integrity of structure and function as is possible; meanwhile preserving the patient from all influences likely to interfere with the process or to set up other mischief, such as cystitis or bedsores, which would entail fresh danger. There is, however, one therapeutic measure that is of unquestionable value in the treatment of the later stages, especially of the cases in which the recurring symptoms suggest a relapsing myelitis as the sequel of injury; and that is the repeated application of a mild "actual cautery" on each side of the spine opposite the affected region. Several applications should be made, with or without an anæsthetic. It is not desirable to lessen the pain by cocaine, because this is likely to interfere with the influence of the proceeding, the beneficial character of which is undoubted.[*] The degenerative sequelæ of injuries to the cord need the same treatment as the similar degenerations that occur apart from traumatic influences. Whenever there is evidence of displacement of the bones, or reason to suspect that the cord is compressed by fractured fragments, or even by products of secondary inflammation outside it, the propriety of trephining the spine needs to be considered. The problem is chiefly surgical, but the fact that it will probably not be necessary to open the dura mater increases the desirability of giving the patient this chance of relief. If needed, the sooner the measure is adopted, when improvement has ceased, the better.

The treatment of the neuralgic condition of spinal pain and tenderness, which so often succeeds injury, is frequently difficult. Counter-irritation is often useful, either by the actual cautery, blisters, iodine, or repeated sinapisms. Of sedatives, Indian hemp is most effective, next to morphia, which should be used as seldom as possible. Hypodermic injections of cocaine may be tried. When all active mischief is over, and the pain has become purely neuralgic, it is often necessary to encourage the patient to neglect it in some degree, and to exert himself in spite of it, while avoiding whatever increases it in considerable degree and for a considerable time. At the same time, extreme care should be observed by all persons who possess the constitutional states above mentioned, predisposing them to myelitis, &c. This is especially necessary when any symptoms persist, and often difficult to secure, except by uncompromising insistence, in those who have been accustomed to a life of active exertion.

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[*] For first directing my attention to the fact, and the evidence supporting it in these cases, I am indebted to Dr. John Anderson.

FUNCTIONAL AND NUTRITIONAL DISEASES.

FUNCTIONAL DISEASES.

Very little is known, though much is heard, of functional diseases of the spinal cord. It is, indeed, open to doubt whether there are any morbid states which can accurately be thus designated; as was pointed out in the introduction, most morbid states thus described are either due to disturbance of cerebral functions, or are the result of changes in the nutrition of the elements of the cord. The deranged function may be an expression of altered nutrition, as it is of altered structure, but such cases can only be termed "functional" by a loose misuse of words. We have an example of the transference of our conceptions of functional derangements from the brain to the cord, in the fact that hysterical paraplegia is often regarded as a functional affection of the cord because the symptoms have the same distribution as those of organic diseases of the cord; but a little consideration will show that, in a case of purely hysterical paraplegia, the morbid functional condition is cerebral; the brain-centres which act on the legs are at fault, but the condition of the functions of the cord itself may be absolutely normal. The spinal motor centres are in a state of inactivity because the related cerebral centres are inactive, but this is no more a diseased condition of the cord than is its corresponding functional state during physiological rest. Hysterical paraplegia will be described, with other palsies of like origin, in the chapter on hysteria—a malady for which the term "functional" is often employed in various ways, as a convenient euphemism.

At the same time, it must be remembered that there is no sharp line of demarcation between functional derangement and nutritional changes. As already mentioned, no functional state can exist without leaving behind it some corresponding change in the finer molecular nutrition of the structures; and if functional derangement of lower structures results from that of higher cerebral centres, and is maintained for long, the change in nutrition that results may be considerable, and may be so definite as to persist even after its cause has ceased to act. Moreover the general enfeeblement of defective nutrition of the nerve-elements is a potent cause of this functional disturbance, and at once facilitates and augments such effects. The process may be observed in many cases not only of hysteria, but also of other derangements of the cerebral functions, and it is seen also, in lower centres, as an effect of pain.

Isolated disturbance of functional centres in the cord does, however, sometimes occur. It is seen in the conditions of inhibition which are designated "reflex paralysis." Such palsies were once thought to be common and persistent, but it has been proved that many, and it is probable that all, cases of considerable and prolonged palsy, formerly thought to be of reflex origin, are due to organic disease, either primary in the cord or secondary to an ascending inflammation of nerves. But transient paralysis sometimes occurs, which cannot be otherwise explained than as an inhibition of a spinal centre, due to peripheral irritation. Such, for instance, is the curious inability to pass urine which sometimes follows an operation on the anus,—the division of a fistula, for instance, or the removal of hæmorrhoids. The inability may continue absolute for several days. Transient weakness of one arm is said sometimes to follow an operation for empyema (Lépine), but the fact that the weakness may be attended by choreoid movements in both arm and leg of one side (Weil) makes it probable that the influence is exerted on a cerebral rather than on a spinal centre. Considerable paraplegia was thought to be sometimes a reflex effect of disease of the bladder, or of a calculus or other organic disease in the kidney, but it is probable that such cases are always of organic nature. Ascending neuritis, reaching the cord, has also been proved to be the cause of lasting symptoms in many cases supposed to be of reflex origin. In one important class of cases in which functional disturbance of the cord is purely secondary to a morbid state of the brain, organic or not, as has been already mentioned, the expression in the limbs of all deranged functions of the brain is through the spinal cord; but such disturbance of the cord is purely secondary, and ceases when the cerebral centres resume their normal state; or, if it is continued longer, this is merely for a brief space of time, until the disturbed structures can regain their normal state. Thus, in idiopathic epilepsy, the convulsion is produced through the agency of the spinal structures, and their over-action may leave them so exhausted that the reflex functions are for a short time abolished. But such conditions cannot be regarded as coming into the category of functional disease of the cord.

The nearest approach to such disease is presented by certain forms of spasmodic disorder, such as transient fixed spasm, catalepsy, and certain forms of tonic spasm, which depend proximately on spinal centres; and by some varieties of neuralgic and other painful affections or dysæsthesiæ, in which the sensory elements of the cord are deranged in action. But we can seldom feel sure that the symptoms in the former class are dependent on a primary derangement of the cord; more often they are clearly secondary to disturbance of cerebral centres, which may indeed coexist, and the cases are better described among the general functional affections of the nervous system; while those of the second class have features and alliances which make it more convenient to describe them among the forms of neuralgia.

In another series of cases, derangement of the motor functions of the spinal cord is secondary to pain, generally that which has such a seat that it is increased by movement; sometimes pain that is not related to movement, but is so intense as to act, by mere severity, on related structures. It may be in the spine itself, in the trunk, or in the limbs. Over-action of the motor centres and spasm may be the result, or the centres may be inhibited, causing a pseudo-paralysis, which may be real, but often blends with a voluntary indisposition to move the parts, lest pain be produced. Spasm is generally brief, and the reflex result of some sudden pain. as is often seen in the case of the "lightning pains" of tabes. When more prolonged contracture results, as in cases in which hysterical contracture follows some painful affection of a limb, the co-operation of cerebral centres cannot be excluded. The inhibitory influence is strikingly seen in the immobility of one half of the thorax in pleurodynia; and in brachial neuritis, the effect may render it impossible to say whether or not there is real motor weakness. Some forms of spasm may perhaps be due to primary disorder of the spinal centres, such as the peculiar cramp in the hands, coming on during sleep, that is described in vol. ii as "nocturnal tetany," and the startings of the limbs on going to sleep; but, for the most part, these spasmodic affections are either secondary to morbid states of the cerebral motor centres, or form part of diseases of wider range in connection with which they are described.

Other cases which may be regarded as functional are those in which symptoms, commonly subjective in character, result from some morbid blood-state. The conditions which most frequently have this effect are gout and diabetes. Occasionally there is definite failure of power, lasting for a few days or weeks, without objective symptoms, and passing away. But the most common symptoms from this cause are sensory and subjective,—feelings of tingling and formication in the legs, dull aching, and sometimes actual pain; this is usually transient, but occasionally continues for some days or weeks, various in position, but, in gouty cases, often felt in the heels. Such symptoms due to morbid blood-states occur chiefly during the second half of life. Those of gouty origin, like other symptoms of the same class, are especially common in persons who inherit a tendency to gout, but have not suffered from attacks of definite arthritis.

In the diagnosis of functional disorders, the first element is the exclusion of any sign of positive disease, and the second is the discovery of some morbid state capable of giving rise to the functional derangement, or of associated symptoms unequivocally functional in character.

The first of these is that of primary and paramount importance, which, alike in practice and reasoning, should precede the others. The various points involved in the diagnosis of hysterical affections are described in the chapter on that disease in vol. ii; one only need be mentioned here—the importance which attaches to the state of myotatic irritability in the legs, and the great difficulty in ascertaining it presented by many of these cases. Permanent excess shows that there is more than functional derangement; although it does not exclude the latter, it indicates changes in nutrition, and will be returned to presently. The cases in which the knee-jerk is apparently absent present a special difficulty. It is never really absent in functional disorder (except when the centres are exhausted by violent convulsion), any more than it is in health. Failure to obtain it is due to the inability of the patient to relax the muscles, which may render attempt after attempt futile, until, in some fortunate moment, relaxation is secured and a characteristic jerk is obtained. The expedients that facilitate the attempt have been already mentioned (p. 15), and the subject has been sufficiently discussed in the account of the diagnosis of tabes.

The treatment of functional disorders is that of the morbid states to which they are due, or of which they form part, and does not need special description. That of the symptoms due to toxic influences is necessarily the removal of the condition which causes them, but they may be to some extent relieved by sedatives, of which bromide of potassium, cocaine, Indian hemp, cimicifuga, and small (7 or 10 gr.) doses of chloralamide are the most effective.

NUTRITIONAL DISEASES.

Among the cases often classed as functional diseases of the spinal cord are some in which objective symptoms of deranged function, slight in degree but definite in character, persist for months, years, or for life. They commence chiefly in those in the early and middle period of adult life, and are more common in women than in men. Such symptoms are inability to walk more than a short distance without fatigue, impaired nutrition of the legs, slight increase of myotatic irritability, often associated with pain in the back, aching in the legs (sometimes amounting to actual pain), and various spontaneous sensations of tingling, "pins and needles," formication, and the like. There is often aching in the legs at night, especially when the patient lies on the back, and hence probably due to passive accumulation of blood in the spinal vessels. Such symptoms occur especially in those of neurotic disposition, who often present other signs of the nervous weakness that it is now fashionable to term "neurasthenia." In many cases, if the history of such symptoms is traced, they will be found to date from some definite exciting cause, from an attack of acute illness, such as typhoid fever or acute rheumatism, from pregnancy, a fall, over-exertion, and the like, or to have come on during a period of general or nerve weakness, anæmia, anxiety, &c. Enfeeblement of the nervous system often results, and the impaired nutrition of the cord may be only part of a similar general state. A slight increase in myotatic irritability is exceedingly common, enough to permit the knee-jerk to be obtained by tapping the depressed patella; but there is rarely a distinct clonus,—at most only two or three jerks, quickly ceasing. We cannot conceive that symptoms so persistent can depend on any mere functional derangement; it is probable that they depend upon changes in the finer nutrition of the nerve-elements, too slight to be detected by the microscope, but causing a corresponding and persistent alteration of function. We have seen that the termination of the upper segment of the motor path is probably that structure of the cord which has least nutritional stability, and is therefore most susceptible of nutritional derangement. Its degeneration is the apparent cause of persistent excess of myotatic irritability, and hence we can understand the frequency with which there is such an excess in these cases. There are probably gradations between such conditions and actual structural disease, in which minute examination reveals visible alteration; and there are certainly gradations between the symptoms above described, and those in which the derangement of function is so considerable and special that the case must be regarded as one in which actual disease exists, of one or other of the types described in the preceding pages. We have already had occasion to consider these nutritional changes as probably underlying the condition of arthritic muscular atrophy, and we have seen that the alterations in spinal nutrition in that disease are apparently the result of the impressions on the peripheral nerves. In women, such a condition is often associated with uterine or ovarian pain, and with sacral pain apparently of uterine origin. It is possible that the condition we have been considering is sometimes secondary to such uterine pain, which acts in a manner somewhat analogous to that in which joint inflammation acts, and that this is the explanation of many of the cases in which a reflex disturbance of the functions of the cord has been supposed to be of uterine origin. The condition is also often associated with spinal pain and tenderness, which may have a similar influence.

In the diagnosis of these nutritional affections of the spinal cord, it is important to remember that they may be closely simulated by analogous changes in the peripheral nerves, constituting the slighter degrees and forms of polyneuritis. The chief points in the distinction have been described in the account of that disease. It is in the parenchymatous forms, and especially in the sensory varieties, that the danger of confusion is apt to arise. Symmetry in the distribution of the subjective sensations, or positive anæsthesia, and their localisation in the extremities, are the most important features of neuritic change.

Connected with this distinction is another, of not less importance. Nutritional changes, equally with the simpler functional derangement, may be the result and expression of the action of some toxic blood-state, and all that has been said in connection with mere alteration in function applies also to the class now under consideration. Toxic influences cannot, indeed, act for long without leading to changes in nutrition, which may speedily attain a degree that amounts to visible structural disease. A search for such an agency should never be neglected in any case in which the symptoms have not followed an adequate cause, and do not form part of a general state of the same nature. But it must be remembered that the peripheral nerves are, as a rule, more susceptible to toxic influences than is the spinal cord, and this fact gives additional weight to any indications that it is from these that the symptoms proceed. Lastly, symptoms suggestive of mere changes in the finer nutrition of the nerve-elements are frequently the earliest indications of organic disease of one or another of the types already described, and in all recent and progressive cases, their general character and aspect must be carefully considered and compared with the known features of the various organic diseases.

The cases with such slight but persistent symptoms as have been just described, vary much in their features, and may present all sorts of slight symptoms, in the utmost diversity of degree and combination. To consider them in detail would be to describe an indefinite series of individual cases, no two of which are identical. The precise degree and manner in which the nerve-elements suffer may, it is evident, be almost infinitely various. Their corresponding manifestations present features, combinations, and gradations, that entirely baffle an attempt to designate them, or even to perceive definite types about which they can be grouped. These are some of the cases of disease which exemplify the failure of types in practical work, the error involved in the attempt to give names, and the paramount importance, in the process of diagnosis, of considering what morbid condition the symptoms indicate, and of dealing with each case as a new problem, *sui generis*, whether or not it is ultimately found to be one of a familiar series. This method, moreover, conducts the practitioner at once to the morbid processes that need treatment, and should determine the character of his efforts to alter that which is at fault.

It is unfortunately not common for the indications of nutritional disturbance of the cord, when they have been long established, to pass away altogether, although some degree of improvement can often be secured; and in many cases, especially in those of brief duration, approximate restoration to a normal state may take place.

The treatment necessarily varies according to the precise condition that exists, and the causes on which it depends. The removal of these as far as possible, and of any influence likely to depress the typhoid fever or acute rheumatism, from pregnancy, a fall, overexertion, and the like, or to have come on during a period of general or nerve weakness, anæmia, anxiety, &c. Enfeeblement of the nervous system often results, and the impaired nutrition of the cord may be only part of a similar general state. A slight increase in myotatic irritability is exceedingly common, enough to permit the knee-jerk to be obtained by tapping the depressed patella; but there is rarely a distinct clonus,—at most only two or three jerks, quickly ceasing. We cannot conceive that symptoms so persistent can depend on any mere functional derangement; it is probable that they depend upon changes in the finer nutrition of the nerve-elements, too slight to be detected by the microscope, but causing a corresponding and persistent alteration of function. We have seen that the termination of the upper segment of the motor path is probably that structure of the cord which has least nutritional stability, and is therefore most susceptible of nutritional derangement. Its degeneration is the apparent cause of persistent excess of myotatic irritability, and hence we can understand the frequency with which there is such an excess in these cases. There are probably gradations between such conditions and actual structural disease, in which minute examination reveals visible alteration; and there are certainly gradations between the symptoms above described, and those in which the derangement of function is so considerable and special that the case must be regarded as one in which actual disease exists, of one or other of the types described in the preceding pages. We have already had occasion to consider these nutritional changes as probably underlying the condition of arthritic muscular atrophy, and we have seen that the alterations in spinal nutrition in that disease are apparently the result of the impressions on the peripheral nerves. In women, such a condition is often associated with uterine or ovarian pain, and with sacral pain apparently of uterine origin. It is possible that the condition we have been considering is sometimes secondary to such uterine pain, which acts in a manner somewhat analogous to that in which joint inflammation acts, and that this is the explanation of many of the cases in which a reflex disturbance of the functions of the cord has been supposed to be of uterine origin. The condition is also often associated with spinal pain and tenderness, which may have a similar influence.

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The treatment necessarily varies according to the precise condition that exists, and the causes on which it depends. The removal of these as far as possible, and of any influence likely to depress the nervous system, must be the first consideration. It should be remembered that no part of the nervous system is unaffected by mental depression, and that impairment of the general health may render impossible recovery from local disease. From these general principles, the details of the treatment of individual cases, too various to be here described, may readily be deduced. Therapeutic measures that are thus reasoned out and determined on by the indications of the special case are far more likely to be successful than those that are simply taken from a description of treatment. The only points that need special mention are, first, the importance of securing mental tranquillity by producing the conviction, when possible, that no grave disease exists or is impending, and with it the disregard of those slighter sensations of discomfort that become more aggressive and disabling the more they are noticed. At the same time, it is important that the patient should avoid all over-exertion, all risk of falls or chills, and should be especially careful in regard to sexual intercourse, which often has a peculiar effect in augmenting the symptoms; and in the unmarried, perfect chastity should be enjoined. Often, indeed, in the case of men, the mental preoccupation causes an apparent failure of sexual desire, which itself is a source of concern—generally groundless. Reassurance on this point frequently helps to secure the desired mental state. Equally important is it to disabuse the sufferer's mind of the idea that seminal loss is weakening—an error widely prevalent; the truth being that any depressing influence comes only from the associated nervous action. But it is important to lessen undue frequency of nocturnal emissions, and for this nothing is so effective as Milton's remedy, large doses of the tinct. ferri perchlor. (♏xx or xxx) three times a day, coupled with the avoidance of lying on the back.

The relief of disabling pain, by the measures described in the chapter on neuralgia, and by the treatment of any local morbid state, is indispensable. Rest from work, the improvement of the general health, and the administration of nervine tonics, such as arsenic, phosphorus, quinine, strychnia, and the like, are the chief other points in treatment. A course of massage, or of gentle faradisation of the muscles, is also often useful.

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